OUTLINE OF ORTHOPAEDICS

BY

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Preface

This book is intended primarily to help students who are studying for the qualifying examinations. I hope that it may also be of use to practitioners whose work brings them into occasional contact with orthopaedic problems, and to physiotherapists and orthopaedic nurses.

My endeavour has been to present an easily read account of our present knowledge and thought on orthopaedic surgery in the shortest possible compass consistent with accuracy, and without resorting to the style of a synopsis. Rarities that are unimportant to the undergraduate student have been omitted, and descriptions of operative technique have been cut down to the barest essentials. Fractures have been excluded because the publishers and I believe that they could be considered more appropriately in a companion volume.

Despite limitations of space I thought it right to include some notes on the methods of examining joints and limbs, because I believe that a clear exposition of clinical methods is the most important contribution that the orthopaedic surgeon can make to the student's surgical training. If the examination candidate can examine a limb competently and elicit the physical signs correctly, his battle is more than half won: and the knowledge will stand him in good stead throughout his clinical career.

To conform with the teaching at most British schools of anatomy the Birmingham revision of the Basle nomenclature has been used throughout this work.

I am grateful to many colleagues for allowing me to use illustrations from their case records. In particular I would like to thank Mr W. Brockbank and Mr D. Ll. Griffiths for Figures 1 and 2, Mr R. M. Handfield-Jones for Figure 65, Mr J. I. P. James for Figure 106,

Mr H. Osmond-Clarke for Figure 63, Dr R. Lightwood for Figures 71 and 72, Mr T. M. Prossor for Figures 50, 55 and 57, and Dr E. Rohan Williams for Figures 43, 44, 73, 76 and 266. Figures 1 and 2 are reproduced by permission of the British Editor and Publishers of the *Journal of Bone and Joint Surgery*.

It is a pleasure to thank Mr Charles Macmillan and Mr James Parker, of Messrs E. & S. Livingstone Ltd., for their encouragement and willing help; and Mr R. W. Matthews, of the same firm, who patiently elaborated the drawings from my rough and inartistic sketches. Many of the illustrations are the work of the photographic department of St Mary's Hospital Medical School, and I am grateful to the director, Dr P. N. Cardew, for his helpful cooperation.

J. C. ADAMS.

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Contents

Introduction

THE term orthopaedic is derived from the Greek words ορθος (straight) and παις (child). It was originally applied to the art of correcting deformities by Nicolas André, a French physician, who in 1741 published a book entitled *Orthopaedia: Or the Art of Correcting and Preventing Deformities in Children: By such Means, as may easily be put in Practice by Parents themselves, and all such as are Employed in Educating Children.*

In André's time orthopaedic surgery in the form known to-day did not exist. Surgery was still primitive. Indeed, except for sporadic attempts by ingenious individuals, it is probable that little real progress had been made since the days of Hippocrates.

That is not to say that surgeons were unintelligent or that they lacked a capacity for careful study and research. Early writings prove that many of them were shrewd observers, and from the time of John Hunter (1728-93) onwards this was increasingly true. Take, for example, the words of Sir Astley Cooper (1768-1848) in his *Treatise on Dislocations and Fractures of the Joints*: "Nothing is known in our profession by guess; and I do not believe, that from the first dawn of medical science to the present moment, a single correct idea has ever emanated from conjecture. It is right, therefore, that those who are studying their profession, should be aware that there is no short road to knowledge; that observations on the diseased living, examinations of the dead, and experiments upon living animals are the only sources of true knowledge; and that inductions from these are the sole basis of legitimate theory."

The enthusiasm and the capacity for study were there. The real obstacle to progress was the lack of the essential facilities that we now take so much for granted—anaesthesia, asepsis, powerful microscopes, and x-rays. Any surgical operation that could not be completed within a few minutes was out of the question when the patient's consciousness could be clouded only by intoxication or exsanguination. And when every major operation was inevitably

available within the span of a single life-time, at the period of the Industrial Revolution.

The first epoch-making advance was the introduction of anaesthesia. The credit for this should be given jointly to Crawford Long, of Athens, Georgia, who was the first to use

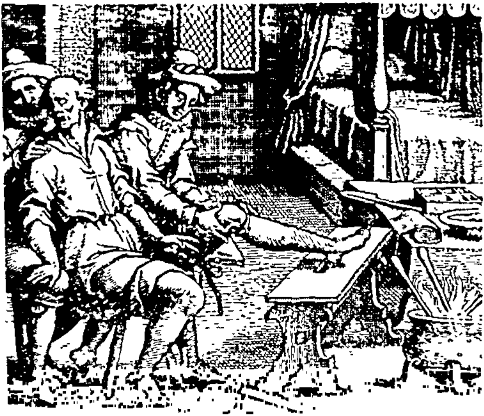

FIG 2

A seventeenth-century amputation scene.
(From Fabricius *Opera*, 1646)

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A few years later Louis Pasteur (1822-95), working in Paris and equipped at last with an adequate microscope, was carrying out his fundamental research on bacteria as a cause of disease. Then in 1867 Joseph Lister (1827-1912), on the basis of Pasteur's work, introduced his antiseptic surgical technique which allowed the surgeon, for the first time in history, to look for primary

followed by suppuration which often proved fatal it is small wonder that operations were seldom advised except in an attempt to save life

Thus orthopaedic surgery, until relatively recent times, was limited to the correction of deformities by rather crude pieces of

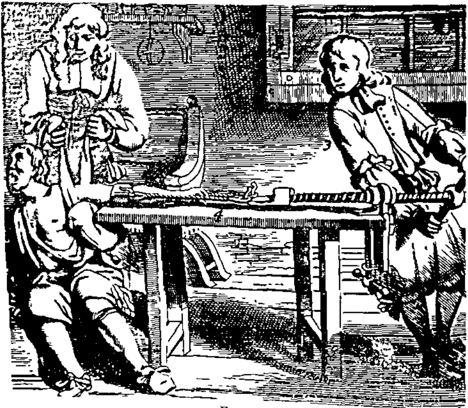

FIG 1

An early method of reducing a dislocated shoulder.
(From Scultetus *Armamentarium Chirurgicum*, 1693)

apparatus, to the reduction of fractures and dislocations by powerful traction (Fig 1), and to amputation of limbs (Fig. 2).

LANDMARKS OF SURGERY IN THE NINETEENTH CENTURY

Fundamental advances in surgery were in fact dependent upon the development of other branches of science and of industry which provided, for instance, the high-powered microscope and the x-ray tube It is therefore not surprising that, after centuries of stagnation, the facilities that were lacking were all made

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healing of his operation wounds. Finally, in 1895, came Roentgen's report from Würzburg in Germany of his discovery of x-rays, which within a short time were put to practical use in surgical diagnosis.

THE EMERGENCE OF ORTHOPAEDICS AS A DISTINCT SPECIALITY

Thus at the dawn of the twentieth century the stage was set for the phenomenally rapid evolution of surgery that has been witnessed by many still alive to-day. With the consequent widening of the scope of surgical practice orthopaedic surgery, at first encompassed by the general surgeon, began to branch off as a distinct science and art; but it was not until after the first world war that it came to be widely recognised as a separate speciality.

In Great Britain many of the fundamental principles of orthopaedics had been propounded, just before the twentieth century began, by Hugh Owen Thomas[1] (1834-91) of Liverpool. But Thomas was not primarily concerned with operative surgery, and it was left to his nephew, Sir Robert Jones (1858-1933) to set orthopaedic surgery upon the sound foundation that it now enjoys. During and after the first world war Robert Jones trained many of the surgeons, British and American, who were among the first to devote their professional lives entirely to the practice of orthopaedics.

To-day the tempo of advance has inevitably slowed, after the first great surge of discovery Yet there remain a great many problems still to be solved, and in this challenge lies the peculiar fascination that orthopaedic surgery holds for its devotees.

THE PRESENT SCOPE OF ORTHOPAEDIC SURGERY

The orthopaedic surgeon is concerned with diseases and injuries of the trunk and limbs His field is not confined to the bones and joints, it includes in addition the muscles, tendons, ligaments, bursae, nerves, and blood-vessels. He is not concerned with injuries of the skull, which fall within the province of the neurosurgeon, or with injuries of the jaws, which are the responsibility of the faciomaxillary or dental surgeon.

[1] Thomas's name is remembered in the widely used Thomas's knee splint and in Thomas's test for fixed flexion at the hip

CHAPTER ONE

Clinical Methods

IN this chapter an attempt will be made to indicate the correct line of approach to an orthopaedic problem, with particular reference to diagnosis and treatment.

As in all branches of medicine and surgery, proficiency in diagnosis can be acquired only from long experience. There is no short cut to a familiarity with physical signs or to skill in radiographic interpretation. Nevertheless the inexperienced surgeon who tackles the problem methodically step by step will often give a better account of himself than his more experienced colleague who makes a " snap " diagnosis after but a cursory investigation.

In the choice of treatment the development of a sound judgment is also largely a matter of experience. Yet more than that is needed. Other essential qualities are common sense and a sympathetic appreciation of human problems. There are surgeons who never acquire a sound judgment however long their apprenticeship. Others seem to have a natural aptitude that quickly matures under proper guidance and training.

THE DIAGNOSIS OF ORTHOPAEDIC DISORDERS

Diagnosis depends first upon an accurate determination of all the abnormal features from 1) the history; 2) clinical examination; 3) radiographic examination; and 4) special investigations. Secondly, it depends upon a correct interpretation of the findings.

HISTORY

In the diagnosis of many orthopaedic conditions the history is of first importance. In cases of torn semilunar cartilage in the knee, for instance, the diagnosis sometimes depends upon the

history alone. Except in the most obvious conditions, a detailed history is always required.

First the exact nature of the patient's complaint is determined. Then the development of the symptoms is traced step by step from their earliest beginning up to the time of the consultation. The patient's own views on the cause of the symptoms are always worth recording: often they prove to be correct. Enquiry is made into activities that have been found to improve the symptoms or make them worse, and into the effect of any previous treatment. Facts that often have an important bearing on the condition are the age and present occupation of the patient, his previous occupations, his hobbies and recreational activities, and previous injuries.

When a detailed history of the local symptoms has been obtained, do not omit to enquire whether there have been symptoms in other parts of the body, and whether the general health is affected. Ask also about previous illnesses.

Finally, in cases that seem trivial, a tactful enquiry why the patient decided to seek advice, and to what extent he is worried by his disability, will often give a valuable clue to the underlying problem. It should be remembered that many patients seek advice not because they are handicapped by their disabilities (which are often insignificant) but because they fear the development of some serious disease such as cancer, tuberculosis, or progressive crippling deformity.

CLINICAL EXAMINATION

The part complained of is examined according to a rigid routine which is never varied. If this is done familiarity with the routine will ensure that no step in the examination is forgotten. Accuracy of observation is essential: it can be acquired only by much practice and by diligent attention to detail.

The examination of the part complained of does not complete the clinical examination. It sometimes happens that symptoms felt in one part have their origin in another. For example, pain in the leg is often caused by a lesion in the spine, and pain in the knee may have its origin in the hip. The possibility of a distant lesion must therefore be considered and an examination made of any region under suspicion.

Finally, localised symptoms may be the first or only manifestation of a generalised or widespread disorder. A brief examination is therefore made of the rest of the body with this possibility in mind.

Thus the clinical examination may be considered under three headings: 1) examination of the part complained of; 2) investigation of possible sources of referred symptoms; and 3) general examination of the body as a whole.

EXAMINATION OF THE PART COMPLAINED OF

The following description of the steps in the clinical examination is intended only as a guide. The technique of examination will naturally be varied according to individual preference. Nevertheless, it is useful to stick to a particular routine, for a familiarity with it will ensure that no step in the examination is forgotten.

Exposure for Examination

It is essential that the part to be examined should be adequately exposed and in a good light. Many mistakes are made simply because the surgeon does not insist upon the removal of enough clothes to allow proper examination.

Inspection

Inspection should be carried out systematically, with attention to the following four points: 1) **The bones:** Observe the general alignment and position of the parts to detect any deformity, shortening, or unusual posture. 2) **The soft tissues:** Observe the soft-tissue contours, comparing the two sides. Note any visible evidence of general or local swelling, or of muscle wasting. 3) **Colour and texture of the skin:** Look for redness, cyanosis, pigmentation, shininess, or other changes. 4) **Scars or sinuses:** If a scar is present, determine from its appearance whether it was caused by operation (linear scar with suture marks), injury (irregular scar), or suppuration (broad, adherent, puckered scar).

Palpation

Again there are four points to consider. 1) **Skin temperature:** By careful comparison of the two sides judge whether there is an area of increased warmth or of unusual coldness. An increase of local temperature denotes increased vascularity. The usual cause is an inflammatory reaction; but it should be remembered that a fast-growing tumour may also bring about a marked local hyperaemia. 2) **The bones:** The general shape and outline of the bones are investigated. Feel in particular for thickening, abnormal prominence, or disturbed relationship of the normal landmarks. 3) **The soft tissues:**

Direct particular attention to the muscles (are they in spasm, or wasted ?), to the joint tissues (is the synovial membrane thickened, or the joint [illegible] made to relate it to a particular structure.

Determining the cause of a diffuse joint swelling. The question often arises : what is the cause of a diffuse swelling of a joint ? The answer can be supplied after careful palpation. For practical purposes a *diffuse* swelling of the joint as a whole can have only three causes : 1) thickening of the bone end ; 2) fluid within the joint ; and 3) thickening of the synovial membrane. In some cases two or all three causes may be combined, but they can always be differentiated by palpation. *Bony thickening* is detected by deep palpation through the soft tissues, the bone outlines being compared on the two sides. A *fluid effusion* generally gives a clear sense of fluctuation between the two hands. *Synovial thickening* gives a characteristic " boggy " sensation—rather as if a layer of soft sponge-rubber had been placed between the skin and the bone. It is nearly always accompanied by a well marked increase of local warmth, for the synovium is a very vascular membrane.

Measurements

Measurement of limb length or circumference is often necessary in the examination of the hip and occasionally of other parts. Details will be given in the chapters on individual regions.

Estimation of Fixed Deformity

Fixed deformity exists when a joint cannot be placed in the neutral (anatomical) position. Its causes are described on p. 26. The degree of fixed deformity at a joint is determined by bringing the joint as near as it will come to the neutral position and measuring the angle by which it falls short.

Movements

In the examination of joint movements information must be obtained on the following points 1) What is the range of active movement ? 2) Is passive movement greater than active ? 3) Is movement painful ? 4) Is movement accompanied by crepitation ?

In measuring the range of movement it is important to know what is the normal With some joints the normal varies considerably from patient to patient , so it is wise always to use the unaffected limb for comparison Limitation of movement in all directions suggests some form of arthritis, whereas selective limitation of movement in some directions with free movement in others is more suggestive of a mechanical derangement

Except in two sets of circumstances passive movement will usually be found equal to the active. The passive range will exceed the active

only in the following conditions: 1) when the muscles responsible for the movement are paralysed; and 2) when the muscles or their tendons are torn, severed or unduly slack.

Power

The power of the muscles responsible for each movement of a joint is determined by instructing the patient to move the joint against the resistance of the examiner. With careful comparison of the two sides it is possible to detect gross impairment of power. By general convention, the strength of a muscle is recorded according to the Medical Research Council grading as follows: 0 = no contraction; 1 = a flicker of contraction; 2 = moderate power, insufficient to move the joint against gravity; 3 = power sufficient to move the joint against gravity; 4 = power to move the joint against gravity plus added resistance; 5 = normal power.

In the occasional instances when more precise information is required muscle strength can be measured against weights, spring balances, or deflection bars.

Stability

The stability of a joint depends partly upon the integrity of its articulating surfaces and partly upon intact ligaments. When a joint is unstable there is abnormal mobility—for instance, lateral mobility in a hinge joint. It is important, when testing for abnormal mobility, to ensure that the muscles controlling the joint are relaxed; for a muscle in strong contraction can often conceal ligamentous instability.

Tests of Function

It is next necessary to test the function of the part under examination. How much does the disorder affect the part in its fulfilment of everyday activities? [illegible] affected. [illegible] is to obse[illegible] Special tests are required to investigate certain functions—for example, the Trendelenburg test for abductor efficiency at the hip (p. 291).

INVESTIGATION OF POSSIBLE SOURCES OF REFERRED SYMPTOMS

When the source of the symptoms is still in doubt after the local examination of the part complained of, attention must be directed to possible extrinsic disorders with referred symptoms. This will entail examination of such other regions of the body as might be responsible. For instance, in a case of pain in the shoulder it might be necessary to examine the neck for evidence of a lesion interfering with the brachial plexus, and the thorax and abdomen for evidence of diaphragmatic irritation, for either of these conditions may be a cause of shoulder pain.

Again, in a case of pain in the thigh the examination will often have to include a study of the spine, abdomen, pelvis, and genito-urinary system as well as a local examination of the hip and thigh.

GENERAL EXAMINATION

The mistake is sometimes made of confining the attention to the patient's immediate symptoms and failing to assess the patient as a whole. It should be made a rule in every case, however trivial it may seem, to form an opinion not only of the patient's general physical condition but also of his psychological outlook. In simple and straightforward cases this general survey may legitimately be brief and rapid. Examine all the limbs for deformity, swelling, wasting, and abnormalities of the joints. Examine the cardio-vascular and respiratory

RADIOGRAPHIC EXAMINATION

The correct interpretation of radiographs becomes easier if the films are examined methodically according to an inflexible routine. In this way abnormalities are far less likely to be missed than they are if one simply gazes hopefully but haphazardly into the viewing box. The following routine is suggested: 1) Set the films in the anatomical position on a viewing box; simply to hold the films up against the light is to invite mistakes. 2) Note what part of the body is shown and by which projections the films have been made. 3) Stand back from the viewing box to assess the *general density* of the bones: judge from experience whether the density seems normal, or whether it is reduced (osteoporosis or rarefaction[1]) or increased (sclerosis). 4) Look more closely for any *local changes of density*. 5) Examine the *cortex* of each bone: run the eye round the outline of the bone, looking for breaks in the continuity of the cortex, and for irregularities or areas of erosion; then examine the substance of the cortex for thickening, thinning, alteration of texture, or new bone formation. 6) Examine the *medulla* of each bone: look for alterations of texture and for areas of destruction or sclerosis. 7) Examine the *joints*: look for narrowing of the "joint" space (more correctly, the cartilage

[1] The term decalcification is inaccurate and should not be used. Calcium is seldom removed from bone without simultaneous absorption of the matrix. This is a cellular process, depending on the activity of osteoclasts.

space), for erosion, irregularity or roughening of the joint surfaces; for peripheral new bone formation (osteophytes), and for loose bodies. 8) Examine the *soft tissues* so far as they are shown: look for relatively dense shadows that might denote abscess or other fluid collection or a solid mass of tissue, and for areas of relative transradiancy that might denote the presence of gas or fatty tissue.

The mistake is often made, when an abnormality has been discovered, of disregarding the rest of the film. It should never be forgotten that two or more separate abnormalities may be present on one film: the routine method of inspection should always be completed regardless of any lesion already discovered. Plenty of time should be spent on the examination of radiographs, and the trap of jumping to hasty conclusions before the films have been properly examined should be avoided at all costs.

Special Radiographic Techniques

In the routine radiography of most parts of the body plain antero-posterior and lateral films are all that are required In cases of difficulty special techniques will give more complete information than the plain films alone.

Tomography. By moving film and x-ray tube in opposite directions during the exposure the structures in the plane corresponding to the axis of movement remain in sharp definition, whereas the structures superficial and deep to that plane are blurred by the movement. By this technique the parts can be shown, as it were, in serial " slices " cut at varying depths from the surface. Tomography is particularly useful in regions such as the spine where, in plain films, the part to be studied is often obscured by overlapping shadows.

Stereoscopic films. These give a three-dimensional picture which is helpful in the study of such regions as the skull, shoulder, spine, and pelvis.

Contrast radiography. In this technique a radio-opaque fluid is injected into cavities or tissue spaces before the radiographs are taken, thereby defining clearly the limits and outline of the space Methods in common use are: *myelography*, to outline the spinal theca; *arthrography*, to outline the cavity of a joint; *arteriography*, to show the arterial tree; and *sinography*, to define

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accurate observation, and a working knowledge of the salient features of the common disorders.

"Functional" or "Psychogenic" Disorders

This heading is included to issue a word of warning. When the cause of a patient's symptoms remains obscure despite a thorough investigation there is a prevalent tendency—it has almost become fashionable—to discount the genuineness of the symptoms and to ascribe them to "hysterical," "functional," or "psychogenic" factors. This must be deplored as a dangerous policy that has led on countless occasions to a serious organic disease being overlooked.

Just because we fail to discover the cause of a particular symptom it by no means follows that the symptom is imaginary; it usually means only that we are not sufficiently skilled in diagnosis. Admittedly, true hysterical disorders are encountered from time to time in orthopaedic practice, but they are few and far between. Much more often a long-continued organic pain leads to a distracted state of mind that is wrongly interpreted as a hysterical manifestation. It is far safer to err on the side of disregarding possible psychogenic factors than to overlook an organic lesion on the supposition that the symptoms are imaginary.

TREATMENT OF ORTHOPAEDIC DISORDERS

Orthopaedic treatment falls into three categories: 1) No treatment—simply reassurance or advice; 2) non-operative treatment; 3) operative treatment. In every case these three possibilities of treatment should be considered one by one in the order given. At least half of the patients attending orthopaedic out-patient clinics (excluding cases of fracture) require no treatment: all that they need is reassurance and advice. In many cases the sole reason for the patient's attendance is that he fears that he may have tuberculosis or cancer. If he can be reassured that there is no evidence of serious disease he goes away satisfied and his symptoms immediately become less disturbing.

If active treatment seems to be required it is a good general principle that whenever practicable a trial should be given first

the course and ramifications of a sinus. In some techniques air can be used as the contrast medium instead of a radio-opaque material.

SPECIAL INVESTIGATIONS

More often than not the diagnosis can be established without having recourse to special investigations. In any case the possibilities should be narrowed down to as few as possible before such investigations are ordered. If doubt then exists, appropriate tests are ordered to support or weaken each possible diagnosis. The tests most commonly employed in orthopaedic diagnosis are: erythrocyte sedimentation rate; haemoglobin estimation and red blood corpuscle counts; leucocyte counts; urine examination; Wassermann or Kahn reaction; gonococcal fixation test; serum calcium estimation; serum inorganic phosphate estimation; alkaline phosphatase estimation; acid phosphatase estimation; blood uric acid estimation; bacteriological examination of fluid or solid specimens; lumbar puncture; sternal puncture; histological examination of excised specimens (biopsy).

INTERPRETATION OF THE FINDINGS

When the study of the patient is complete the abnormal findings elicited from the history, clinical examination, radiographic examination, and appropriate special investigations should be assembled together to form a composite clinical picture. This can then be matched against the recognised disorders of the region under consideration It is comforting to remember that the number of disorders that commonly affect a particular region is limited Often the number is not large. Theoretically, therefore, if all the possibilities are listed and thereafter confirmed or eliminated one by one the correct diagnosis must always be revealed.

In practice, of course, diagnosis is not so simple as that. But it is nevertheless true that if the problem is tackled logically, step by step, in the manner described, a correct conclusion can be formed in the great majority of cases The only essentials are a capacity for painstaking enquiry, with strict attention to detail,

Half-hearted treatment at infrequent intervals is a waste of time. Ideally it should be practised daily. The following are the most important forms in which physiotherapy is given.

Active exercises. Exercises are given for two different purposes: 1) to mobilise joints, and 2) to strengthen muscles. In *mobilising exercises* the patient's active efforts to move the joint may be assisted by gentle pressure by the physiotherapist's hand (assisted active exercises). In *muscle-strengthening exercises* the patient is encouraged to contract the weakened muscles against the resistance of weights or springs, the resistance being increased as the muscles gain power.

Passive joint movements. These are of limited value. Their chief use is in preserving the range of joint movements when the patient is unable to move the joint actively—that is, when the muscles are paralysed or severed.

Electrical stimulation of muscles. Compared with active exercises this has only a small part to play in strengthening a muscle. If the nerve supply of the weakened muscle is intact, electrical stimulation is applied through the nerve by faradic current (that is, current induced by the make-and-break of an induction coil, or by a special electronic circuit). If the nerve is paralysed the muscle can be stimulated only by the application of an interrupted direct current.

Local heat. Possibly by increasing the blood flow, or possibly in other ways, heat produces a soothing effect on many aching pains of "rheumatic" or "fibrositic" type, although the effect is usually only short-lived. *Surface heat* is applied by an infra-red or radiant-heat lamp. *Deep heat* is applied by short-wave diathermy, which produces the greatest heat at a point between the two electrodes.

Massage. Massage has only a limited field of usefulness. In general, it is far less effective than active exercises in improving the circulatory return in cases of gravitational oedema. Nevertheless it is occasionally useful as a supplementary measure in such cases; and it may also be of value in loosening subcutaneous scars.

Local Injections

In this category are included injection of local anaesthetic solutions (occasionally other compounds) into areas of tenderness

to non-operative measures. Most orthopaedic operations fall into the category of "luxury" rather than life-saving procedures. Consequently the patient should seldom be persuaded to submit himself to operation: rather should he have to bully the surgeon into performing it. When one is undecided whether to advise conservative or operative treatment it is wise always to err on the side of non-intervention.

METHODS OF NON-OPERATIVE TREATMENT

Rest

Since the days of H. O. Thomas, who emphasised its value in diseases of the spine and limbs, rest has been one of the mainstays of orthopaedic treatment. Complete rest demands recumbency in bed or immobilisation of the diseased part in plaster. But by "rest" the orthopaedic surgeon does not necessarily mean complete inactivity or immobility. Often he means no more than "relative rest," implying simply a reduction of wonted activity and avoidance of strain.

Support

Rest and support often go together; but there are occasions when support is needed but not rest—for example, to protect a joint rendered insecure by muscle paralysis. When support is to be temporary it can be provided by a plaster case or splint. When it is to be prolonged or permanent an individually made surgical appliance is required. Examples in common use are steel-reinforced lumbar corsets, spinal braces, walking calipers, below-knee steels with ankle straps, and drop-foot springs.

Physiotherapy

Physiotherapy in its various forms occupies an important place in the non-operative—and in the post-operative—treatment of orthopaedic disabilities. Being easily prescribed, and entailing no trouble to the surgeon, it is no doubt often misused: much treatment is given that can have no beneficial effect except perhaps psychologically. Nevertheless in appropriate cases appropriate physiotherapy is of real value. Especially is this so in the rehabilitation of patients after injury or operation.

When it is used, physiotherapy should be pursued thoroughly.

the joint or, more often, in the soft tissues near the joint. Before undertaking manipulation for joint stiffness the surgeon must make sure that the joint is free from active infection or inflammatory disease, which would almost certainly be worsened by manipulation. For this reason radiographs must always be obtained before manipulation is considered. Great force should not be used: it is better to gain slight improvement by repeated gentle manipulation than to rely upon a single forcible manoeuvre. Excessive force may fracture a bone or cause fresh bleeding within the joint, thereby aggravating the stiffness. Manipulation for joint stiffness should always be followed by a course of active exercises designed to retain the increased range of movement.

Manipulation for correction of deformity. This has its most obvious application in the reduction of fractures and dislocations. It is also used to overcome spontaneous deformities from spastic muscles or contracted soft tissues—as, for example, in congenital club foot. Repeated manipulation may be necessary, with or without anaesthetic, a little further improvement being gained each time. After manipulation for correction of deformity the limb is usually immobilised on a splint or in plaster to maintain the correction.

Deep X-ray Therapy

X-ray therapy is the mainstay of treatment in ankylosing spondylitis, in certain cases of osteoclastoma, and in malignant disease when more radical treatment is impracticable.

OPERATIVE TREATMENT

The chief essential of any operation is that it should not make the patient worse than he was before he submitted himself to it. This is so obvious that the statement may almost sound absurd. Yet it is unfortunately true that a disturbing number of operations carried out for orthopaedic conditions do in fact cause more harm than good for one reason or another. Hence the selection of cases for operation, the choice of the most appropriate operation in given circumstances, the technical performance of the operation, and the post-operative management are matters of the highest importance, and they call for a high degree of judgment and skill. Herein lies much of the fascination of orthopaedic surgery.

or into joints. Temporary relief may sometimes be afforded. The method is possibly worth a trial in selected cases, in that it can seldom do harm; but one should not place too much reliance upon it.

Drugs

Drugs have rather a small place in orthopaedic practice. Those used may be placed in three categories: 1) antibiotics; 2) analgesics; and 3) specific drugs. *Antibiotics* are of immense importance in infective lesions, especially in acute osteomyelitis and acute pyogenic arthritis; but to be successful treatment must be begun very early. Antibiotics are also of definite value in certain chronic infections, notably in tuberculosis. *Analgesics* should be used as little as possible. Many orthopaedic disorders are prolonged for many weeks or months, and it is undesirable to prescribe any but the mildest analgesics continuously over long periods. *Specific drugs* work well in certain special diseases. Examples are vitamin C for scurvy, vitamin D for rickets, colchicum for gouty arthritis, salicylates for the arthritis of rheumatic fever, and stilboestrol for the metastatic deposits of carcinoma of the prostate.

Manipulation

The merits and dangers of manipulation as a method of treatment form a frequent topic of controversial discussion. On the one hand are the unqualified practitioners who claim to relieve all manner of diseases by manipulative replacement of a "displaced" bone On the other are the practical surgeons who, sceptical of its value, confine manipulation to a narrow field. Certainly there is no rational justification for the activities of the indiscriminate manipulator, who usually shows a total disregard for pathology But equally there is no doubt that, with proper precautions and in appropriate conditions, manipulation has its place in treatment.

Broadly, the indications for manipulation fall into two groups. It may be undertaken 1) to restore movement to a stiff joint; or 2) to overcome deformity.

Manipulation for joint stiffness. This is seldom required. It finds its greatest usefulness in cases of joint stiffness after injury. In such cases stiffness is usually caused by adhesions, either within

extra-articular, or the two may be combined. In *intra-articular* arthrodesis the joint is opened and the bone ends are displayed. The articular cartilage (or what remains of it) is removed so that raw bone is exposed. The joint is placed in the desired position and immobilised until clinical tests and radiographs show sound bony fusion.

In *extra-articular* arthrodesis the joint itself is left undisturbed, but it is "by-passed" by securing bone-to-bone fusion above or below the joint, usually through the medium of a bone graft. The method is applicable mainly to the spine, shoulder, and hip. It has a theoretical advantage in cases of infective joint disease, because any risk of reactivating or disseminating the infection by opening the joint is avoided.

Examples of the methods of arthrodesing the shoulder and hip are illustrated in Figures 3 and 4.

Position for arthrodesis. The best position for arthrodesis should not be regarded as rigidly established for each joint. Variations may be appropriate and desirable in individual cases—for instance, to conform to the requirements of the patient's work. The following is simply a general guide. *Shoulder :* About 30 to 40 degrees of abduction, 20 degrees of flexion, and 20 degrees of medial rotation *Elbow :* If only one elbow is affected, 90 degrees of flexion (or according to the requirements of the patient's work). If both elbows are affected one should be in flexion above the right angle and the other about 20 degrees below the right angle. If forearm rotation is lost the most useful position of the forearm is in 10 degrees of pronation. *Wrist :* Dorsiflexion of 20 degrees. *Interphalangeal joints :* Semiflexed. *Hip :* Neutral position (not more than 15 degrees of flexion). *Knee :* About 20 degrees of flexion. *Ankle :* In men, right angle ; in women, 20 to 30 degrees of plantar flexion, according to accustomed height of heel.

Arthroplasty

Arthroplasty is the operation for construction of a new movable joint It has a much narrower field than arthrodesis. In practice, its use is confined mainly to the elbow, the hip, the knee, and the metacarpo-phalangeal and metatarso-phalangeal joints.

Indications. The indications for arthroplasty are not well defined, for there is considerable diversity of opinion among different

A detailed account of operative techniques is unnecessary here. All that is required is a brief mention of the more important operations.

Arthrodesis

The operation of arthrodesis, or joint fusion, has a wide application. The disability from a single stiff joint is slight, and patients readily adapt themselves to it. Even when two or three

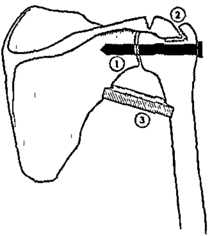

FIG. 3
Three methods of arthrodesis of the shoulder 1. Intra-articular arthrodesis with fixation by a nail 2 Extra-articular arthrodesis, acromion turned down into a slot in the greater tuberosity. 3 Extra-articular arthrodesis · strut graft between humerus and scapula

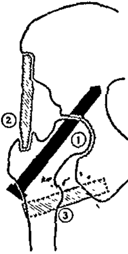

FIG. 4
Three methods of arthrodesis of the hip. 1. Intra-articular arthrodesis with fixation by a nail 2. Extra-articular arthrodesis by ilio-femoral graft. 3 Extra-articular arthrodesis by ischio-femoral graft

joints are fused function may be surprisingly good, depending upon the particular joints affected

Indications. Arthrodesis is indicated mainly in the following conditions: 1) Advanced osteoarthritis with disabling pain, especially when confined to a single joint 2) Quiescent tuberculous arthritis with destruction of the joint surfaces, to eliminate risk of recrudescence and to prevent deformity. 3) Instability from muscle paralysis, as after poliomyelitis 4) For permanent correction of deformity, as in "hammer" toe.

Methods of arthrodesis. Arthrodesis may be intra-articular or

extra-articular, or the two may be combined. In *intra-articular* arthrodesis the joint is opened and the bone ends are displayed. The articular cartilage (or what remains of it) is removed so that raw bone is exposed. The joint is placed in the desired position and immobilised until clinical tests and radiographs show sound bony fusion.

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Indications. The indications for arthroplasty are not well defined, for there is considerable diversity of opinion among different

surgeons Broadly, it has a use in the following conditions: 1) advanced osteoarthritis with disabling pain, especially in the elbow, hip, and metatarso-phalangeal joints; 2) for the correction of certain types of deformity (especially hallux valgus); 3) quiescent tuberculous arthritis of the elbow, with destruction of the joint surfaces; 4) certain ununited fractures of the neck of the femur. It will be realised that in several of these conditions arthroplasty is an alternative to arthrodesis.

Methods of arthroplasty. Three methods are in general use. Each has its merits and disadvantages.

Excision arthroplasty. In this method one or both of the articular ends of the bones are simply excised, so that a gap is created

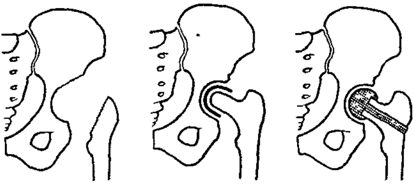

FIG 5 FIG 6 FIG 7

Three methods of arthroplasty, as exemplified at the hip. Figure 5—Excision arthroplasty. Figure 6—Cup arthroplasty Figure 7—Replacement arthroplasty

between them (Fig 5) The gap fills with fibrous tissue, or a pad of muscle or other soft tissue may be sewn in between the bones By virtue of its flexibility the interposed tissue allows a reasonable range of movement, but the joint often lacks stability. The method is applicable to all the joints for which arthroplasty is practicable except the knee It is used most commonly at the metatarso-phalangeal joint of the great toe, in the treatment of hallux valgus and hallux rigidus

Cup arthroplasty This method lends itself particularly well to ball-and-socket joints In practice its use is virtually limited to the hip The joint surfaces are refashioned to a true hemispherical form, and a highly polished cup of inert metal or plastic is interposed between them as a new "lining" (Fig. 6). Movement

occurs at both surfaces of the cup, no attempt being made to fix it immovably to either joint surface. In the course of time the constant friction of the bones against the polished surface of the cup stimulates the development of a surface-layer of smooth fibrocartilage. A serious disadvantage of this method is that the blood supply of the bone within the cup is in jeopardy.

Replacement arthroplasty. This method is still in a stage of development. In principle one (or sometimes both) of the articulating bone ends is excised and replaced by an inert prosthesis of similar shape (Fig. 7). Ideally the prosthesis should remain rigidly fixed to the stump of bone to which it is fitted, but in practice some loosening is probably inevitable. First developed for use in the hip (for ununited fractures of the femoral neck), the method has been adapted, with various modifications, to other joints such as the knee and the metacarpo-phalangeal joints.

Bone Grafting Operations

Bone grafts are usually obtained from another part of the patient's body (autogenous grafts). If it is impracticable or undesirable to take bone from the patient's own body, grafts from another human subject may be used (homogenous grafts). They are generally stored frozen in a " bone bank " until required. Grafts obtained from animals (heterogenous grafts) are not recommended.

Bone transferred as a graft from one site to another does not survive wholly in a living state For the most part the bone cells die, although a few that are near the surface may possibly survive. The purpose of the graft is mainly to serve as a scaffolding or temporary bridge upon which new

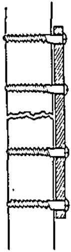

FIG 8

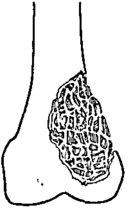

FIG. 9

in a bone.

bone is laid down. Thus the whole of a graft is eventually replaced by new living bone.

Indications. Bone grafts are used mainly in three types of case: 1) in cases of ununited fracture, to promote union; 2) in arthrodesis of joints, either to supplement an intra-articular arthrodesis or to promote extra-articular fusion; 3) to fill a defect or cavity in a bone.

Technique. Two basic techniques are in general use. In one, bone is transferred in the form of a solid slab; in the other, as small fragments or chips.

Slab grafts. These are usually obtained from strong cortical bone: the subcutaneous part of the tibia is a common site. The graft is fixed to the recipient bone either by screws or by inlaying. Such a graft serves as an internal splint as well as a framework for the growth of new bone (Fig. 8).

Chip grafts. These are obtained from spongy cancellous bone. The crest of the ilium is the usual donor site. The chips are packed firmly into, or around, the recipient bone and are held in place simply by suture of the soft tissues over them (Fig. 9).

Tendon Transfer Operations

In the operation of tendon transfer, or tendon transplant, the insertion of a healthy functioning muscle is moved to a new site, so that the muscle henceforth has a different action. In this way the function of a paralysed or severed muscle can be taken over by one that is intact In properly selected cases there need be no noticeable loss of power in the former sphere of action of the transplanted muscle, for there is often considerable duplication or overlap in the function of individual muscles. Thus a tendon of the flexor digitorum sublimis may be transferred to a new site without appreciably impairing the power of finger flexion, which can be adequately controlled by the flexor profundus. Similarly the extensor indicis can be spared for a new function without seriously interfering with the power of extension of the finger

Indications. Tendon transfers have their main application in three groups of conditions: 1) in cases of muscle paralysis, to restore or improve active control of a joint; 2) in cases of ruptured or cut tendon, when direct suture of the ends is impracticable;

and 3) in cases of deformity from muscle imbalance, to maintain correction.

Technique. The tendon to be transplanted is divided at an appropriate point, re-routed in the direction of its new action, and secured by stainless steel sutures to its new insertion (Fig. 186, p. 273). If it is to be inserted into bone it is passed through a drill hole and held by suturing back on to itself. If it is to be united to a tendon stump the junction may be secured by end-to-end suture or, preferably, by interlacing the tendons one through the other and transfixing them with mattress sutures.

Tendon Grafting Operations

In tendon grafting a length of free tendon is used to bridge a gap between the severed ends of the recipient tendon.

Indications. The chief use of free tendon grafts is in the reconstruction of flexor tendons severed in the fibrous digital sheaths of the hand (p. 274).

Technique. The free tendon graft is usually obtained from the palmaris longus or from one of the toe extensors at the dorsum of the foot. Proximally, it is joined to the recipient tendon by sutures of stainless steel wire. Distally, it may be secured to the distal stump of the recipient tendon or it may be attached directly to bone through a drill hole.

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CHAPTER TWO

General Survey of Orthopaedic Disorders

THIS chapter is devoted to a broad preliminary review of the field of orthopaedics. The main groups of disorders will be described without a detailed consideration of their local manifestations. Against this general background the features of the common disorders as they affect each particular region will be discussed in the subsequent chapters.

Classification

Most orthopaedic disorders fall within the following groups:

INJURIES

DEFORMITIES

- Congenital deformities
- Acquired deformities

AFFECTIONS OF JOINTS

- Arthritis
- Dislocation and subluxation
- Internal derangements
- Other mechanical derangements

AFFECTIONS OF BONE

- Infections of bone
- Tumours of bone
- Other local affections of bone
- General affections of the skeleton

AFFECTIONS OF SOFT TISSUE

- Inflammatory lesions of soft tissue
- Tumours of soft tissue

NEUROLOGICAL DISORDERS

- Poliomyelitis
- Spastic paralysis
- Peripheral nerve lesions

INJURIES

Recent injuries of the limbs and spine form a subject for special study, and they are dealt with in textbooks on fractures. Injuries will be considered here only in so far as they contribute to persistent or recurrent disability.

CONGENITAL DEFORMITIES

Congenital abnormalities of the limbs and trunk take many different forms. Only three anomalies are of major importance to the orthopaedic surgeon—congenital dislocation of the hip (p. 293), congenital club foot (p. 378), and cervical rib (p. 137). Most of the remainder have little practical importance and the student should not burden his mind by studying them in needless detail.

Classification

Congenital abnormalities can be classified into three broad groups: 1) absence of a part; 2) defect of a part; and 3) development of a supernumerary part (Figs. 10-12).

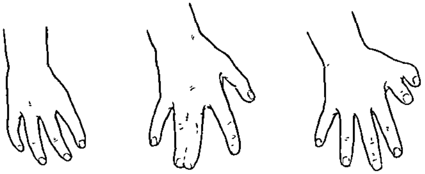

FIG. 10 FIG. 11 FIG. 12

Three types of congenital deformity, exemplified in the hand. Figure 10—Absence of a part. Figure 11—Defect of a part. Figure 12—Development of a supernumerary part

ABSENCE OF A PART

Anything from a whole limb to the tips of the digits may be congenitally absent (Fig. 10). Sometimes one element of a limb alone is absent—for example, the radius, the clavicle, the upper half of the femur, or the sternomastoid muscle.

DEFECT OF A PART

In this group all the main elements are present, but some or all fail to develop normally (Fig. 11). It is the largest group. It includes such abnormalities as achondroplasia (p. 86), syndactylism, congenital dislocation of the hip (p. 293), and congenital club foot (p. 378).

DEVELOPMENT OF A SUPERNUMERARY PART

This group includes the fairly common condition of supernumerary digit (Fig. 12), cervical rib (p. 137), and the rare deformities in which a teratomatous mass, perhaps representing an ill-formed twin, is attached to the body, usually in the sacral region.

ACQUIRED DEFORMITIES

Acquired deformities are more frequent than congenital deformities. They may be classified in two groups: those in which deformity arises at a joint, and those in which it arises in a bone.

DEFORMITY ARISING AT A JOINT

Deformity may be said to exist at a joint when it cannot be placed voluntarily in the neutral anatomical position.

Causes

The causes of deformity arising at a joint may be summarised as follows (Fig 13):

Dislocation or subluxation. This is usually caused by injury, but it may occur as a congenital deformity, or it may follow disease of the joint (pathological dislocation)

Muscle imbalance. Unbalanced action of muscles upon a joint may hold it continuously in a particular arc of its range In time, secondary contractures occur in the dominant muscles or in the soft tissues, preventing the joint from returning to the neutral position (Fig. 13 (2)). The two fundamental causes of muscle imbalance are: 1) weakness or paralysis of muscles, and 2) spasticity of muscles. Thus equinus deformity at the ankle may

follow paralysis of the dorsiflexor muscles (for instance, in poliomyelitis) because the action of the plantar flexors and of gravity is unopposed. Or a similar deformity may be caused by spasticity of the calf muscles, which overpower their antagonists. This occurs commonly in spastic paralysis (p. 116).

Tethering or contracture of muscles or tendons. If something happens to muscles or tendons that prevents their normal to-and-fro gliding, or their elongation and retraction, the joint

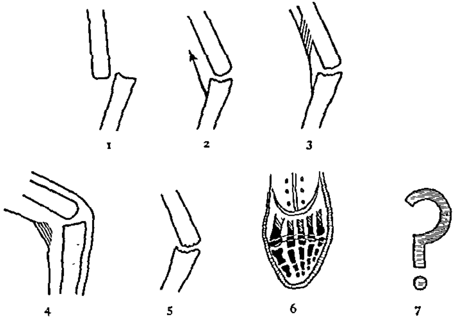

FIG. 13

Seven causes of deformity arising at a joint. 1 Dislocation. 2 Muscle imbalance. 3. Tethering of muscle or tendon. 4. Soft-tissue contracture. 5. *Arthritis.* 6. Posture 7 Idiopathic (cause unknown)

may be held in a position of deformity. Thus a muscle or tendon may be tethered to the surrounding tissues as a result of local infection or injury (Fig. 13 (3)). An example is the anchoring of a flexor tendon of a finger within its fibrous sheath as a result of suppurative tenosynovitis, with consequent flexion deformity of the interphalangeal joints. Or a muscle may lose its elasticity and contractile power as a result of an impaired blood supply. An important example is Volkmann's ischaemic contracture of the forearm flexor muscles (p. 246) from occlusion of the brachial artery near the elbow, causing flexion deformity of the wrist and fingers.

Contracture of soft tissues. Apart from any disturbance of the muscles, contracture of soft tissues alone can account for joint deformity. An example is the common condition of Dupuytren's contracture (p. 269), in which the thickened and contracted palmar fascia pulls the metacarpo-phalangeal and proximal interphalangeal joints of one or more fingers into flexion. Similarly, a flexion deformity of the knee or elbow may occur from contracture of the scarred skin after burns of the flexor surface of the limb (Fig. 13 (4)).

Arthritis. The various types of arthritis will be discussed in a later section of this chapter. Any type of arthritis may lead to joint deformity. In some cases the joint is firmly fixed in a deformed position by bony or fibrous ankylosis. In other instances the joint retains some movement but is prevented from reaching the neutral position. Thus flexion and adduction deformity is common in osteoarthritis of the hip, flexion deformity is common in arthritis of the knee, and the deformity of ulnar deviation of the fingers (Fig. 167, p. 253) is a well known feature of rheumatoid arthritis of the metacarpo-phalangeal joints

Posture. The habitual adoption of a deformed position of a joint often leads in time to permanent deformity. A common example is the lateral deviation of the great toe at the metatarso-phalangeal joint—*hallux valgus*—so common in women who cramp their feet into narrow pointed shoes (Fig. 13 (6)). Another common postural deformity—but one that should never be allowed to occur—is fixed flexion deformity of the knees in patients confined to bed for long periods with the knees bent over a pillow

Unknown causes. In some cases deformity occurs at a joint for no apparent reason Thus many children develop knock-knee deformity between the ages of 3 and 5 years without demonstrable cause It is usually unimportant because it tends to correct itself spontaneously A more sinister deformity that is equally ill-explained is the idiopathic scoliosis of adolescents (p. 154)

DEFORMITY ARISING IN A BONE

Deformity exists in a bone when it is out of its normal anatomical alignment.

Causes

There are three causes of deformity arising in bone (Fig. 14).
Fracture. This is by far the most common cause. Unless a fracture is reduced so that the fragments are perfectly aligned deformity will result. Examples are the genu valgum (knock-knee) that is often the consequence of compression fractures of the lateral tuberosity of the tibia, the cubitus valgus that may follow displaced fractures of the lateral condyle of the humerus, and the common "dinner-fork" deformity of an unreduced fracture of the lower end of the radius.

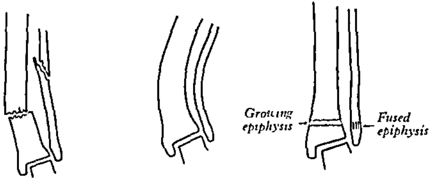

FIG 14

Three causes of deformity arising in a bone. 1 Fracture. 2. Bending of softened bone 3 Uneven epiphysial growth.

Bending of softened bone. Many unrelated conditions can cause softening of bone, with liability to bending and consequent deformity. They are mostly generalised affections in which several or all of the bones are affected. The following are examples: *Metabolic disorders:* Rickets, osteomalacia. *Endocrine disturbances:* Parathyroid osteodystrophy, Cushing's syndrome. *Affections of unknown cause:* Paget's disease (osteitis deformans), fibrous dysplasia of bone, senile osteoporosis The main features of these affections will be described later in this chapter.

Uneven growth of bone. In children any disturbance of the growing epiphysial cartilage may lead to uneven growth and consequent deformity. The usual effect of interference with a growing epiphysial cartilage is that its growth is retarded; occasionally it is accelerated Deformity will follow only if the growing cartilage is affected more in one part than another, or if

the interference with growth affects one bone of a pair, as in the forearm or leg (Fig. 14, (3)). The most frequent causes of retarded epiphysial growth are: 1) fracture crushing the epiphysial cartilage; 2) infection of the cartilage, usually from adjacent osteomyelitis or joint infection; 3) enchondroma (a benign tumour) adjacent to the cartilage. In the relatively uncommon cases in which epiphysial growth is accelerated the usual cause is local hyperaemia induced by an adjacent focus of infection or by a vascular tumour such as a haemangioma.

Treatment of Deformities

Each deformity must be considered as an individual problem. Many do not require treatment, or are not amenable to it. In other cases an attempt may be made to correct or improve the deformity. One or more of the following methods may be used in appropriate cases: 1) manipulative correction and retention in a plaster or splint; 2) gradual correction by prolonged traction; 3) division or freeing of contracted or tethered soft tissues; 4) osteotomy; 5) selective retardation of epiphysial growth (in children).

ARTHRITIS

The term arthritis is used here to include both inflammatory and degenerative lesions of a joint It implies a diffuse lesion affecting the joint as a whole. It does not include localised mechanical disorders such as loose body formation or tears of the semilunar cartilages, which are better designated as internal derangements Nor should it embrace acute injuries of joints

Clinically, arthritis is generally characterised by pain and restriction of movement at a joint of spontaneous onset, in superficial joints these features are usually accompanied by obvious swelling If a joint is not swollen and if it moves freely and painlessly through its normal range it is highly unlikely that it is affected by arthritis

Types of Arthritis

If rare variations are excluded, there are four common types of arthritis and five uncommon types, making nine types in all. The four common types are 1) pyogenic arthritis; 2) rheumatoid arthritis; 3) tuberculous arthritis; and 4) osteoarthritis.

The five uncommon types are : 1) gouty arthritis ; 2) haemophilic arthritis ; 3) Charcot's osteoarthropathy ; 4) the arthritis of rheumatic fever [1] ; and 5) ankylosing spondylitis.

PYOGENIC ARTHRITIS

(Infective arthritis ; septic arthritis)

In this form of arthritis a joint is infected by organisms of one of the pyogenic groups. Typically there is acute joint infection of rapid development, but the infection may be subacute or even chronic. When pus is formed within the joint the condition is sometimes termed *suppurative arthritis*.

Cause. Staphylococci, streptococci, pneumococci, or gonococci are the organisms usually responsible.

Pathology. The organisms may reach the joint by three routes : 1) through the blood stream (haematogenous infection) ; 2) through a penetrating wound ; or 3) by extension from an adjacent focus of osteomyelitis—especially when the infected metaphysis is wholly or partly within the joint cavity (as are the upper humeral metaphysis, all the metaphyses at the elbow, and the upper and lower metaphyses of the femur (Fig. 31, p. 56)).

The infection causes an acute or subacute inflammatory reaction in the joint tissues. There is exudation of fluid within the joint : the fluid is turbid or frankly purulent according to the severity of the infection. The outcome varies from complete resolution, with normal function, to total destruction of the joint and fibrous or bony ankylosis (Fig. 15).

Clinical features. The onset is acute or subacute, with pain and swelling of the joint There is constitutional illness, with pyrexia. *On examination* the joint is swollen, partly from fluid effusion and partly from thickening of the synovial membrane. When the affected joint is superficial, the overlying skin is warmer than normal and it is often reddened. All movements are restricted ; in severe cases they are almost totally prevented by protective muscle spasm Attempted or forced movement increases the pain A boil or other primary focus of infection is often to be found elsewhere in the body. *Radiographs* show no alteration from the normal in the early stages (Fig. 16). Later, if the infection

[1] Though it is seldom seen in orthopaedic practice, the arthritis of rheumatic fever is encountered more frequently in medical and paediatric clinics.

persists, there may be diffuse osteoporosis, loss of cartilage space, and possibly destruction of bone (Fig. 17). *Investigations:* There

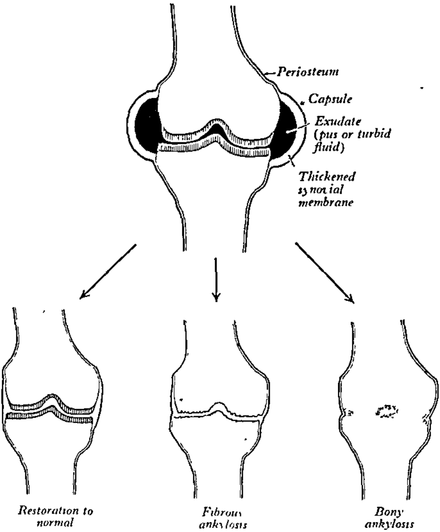

FIG. 15

Pyogenic arthritis, with possible results. In the active phase the joint is distended with pus or turbid fluid, the synovial membrane is inflamed and moderately thickened. The outcome varies with the intensity of the infection and the response to treatment. There may be 1) restoration to normal; 2) fibrous ankylosis; or 3) bony ankylosis.

is a polymorphonuclear leucocytosis. The erythrocyte sedimentation rate is raised. Bacteriological examination of aspirated fluid usually reveals the identity of the causative organism.

Diagnosis. This is from other forms of arthritis (especially tuberculous arthritis, gouty arthritis, and haemophilic arthritis), and from infections near the joint (especially acute osteomyelitis). The rapid onset, pyrexia, leucocytosis, and the character of the aspirated fluid are important diagnostic features.

Fig. 16

Fig. 17

Pyogenic arthritis of the wrist F... abnormality Figure 17—Four ... the slight but significant narrowi... of articular cartila...

Prognosis. This varies widely according to the severity of the infection, the organism responsible, and the promptness with which efficient treatment is begun. Many joints can be saved intact, but many are destroyed more or less completely, with fibrous or bony ankylosis (Fig. 15). A few patients die.

Treatment. Early treatment is absolutely essential if there is to be a reasonable prospect of preserving normal joint function. *Constitutional treatment:* This is by rest in bed, and chemotherapy with appropriate antibiotic drugs. Whenever possible the causative organism must be identified and its sensitivity to antibiotics determined, so that the most effective drug can be given.

Until that information is available treatment should be begun with penicillin in large doses. *Local treatment :* The joint is rested, usually in a plaster splint. The fluid exudate, which is often purulent, is removed by aspiration (or, if necessary, by incision). At the same time a solution of the appropriate antibiotic drug is injected into the joint. Aspiration and injection of antibiotic solutions are repeated daily so long as the exudate continues to form. Rest is enforced until the infection is overcome, as shown by the subsidence of pyrexia and retrogression of the local signs Thereafter active movements are encouraged in order to restore the greatest possible function to the joint.

RHEUMATOID ARTHRITIS

(Rheumatoid polyarthritis)

Rheumatoid arthritis is a chronic non-bacterial inflammation of joints, often associated with mild constitutional symptoms. It nearly always affects several joints at the same time (polyarthritis). Joint changes of a similar nature also occur in certain medical conditions such as Still's disease of children, Reiter's syndrome, psoriasis, lupus erythematosus, and other collagen diseases.

Cause. This is unknown.

Pathology. The synovial membrane is thickened by chronic inflammatory changes (Fig. 18) Much later the articular cartilage is softened and eroded, and in long-established cases there may be small erosions of the bone ends. After months or years of activity the disease " burns itself out," leaving a joint that is usually permanently damaged.

Clinical features. The patient is usually a young or middle-aged adult Any joint may be affected, but the incidence is higher in the more peripheral joints such as the knees, ankles, elbows, wrists, and hands than in the spine, shoulders, or hips. The onset is gradual, with increasing pain and swelling of a joint. Very soon a number of other joints are similarly affected. Pain and stiffness are worst when activity is resumed after resting *On examination* the affected joints are swollen from synovial thickening. The overlying skin is warmer than normal The range of joint movements is limited, and movement causes pain, especially at the extremes. *Radiographic examination :* At first there is no alteration from the normal. Later, there is diffuse osteoporosis

in the area of the joint. Eventually destruction of joint cartilage may lead to narrowing of the cartilage space (Fig. 19) and, in

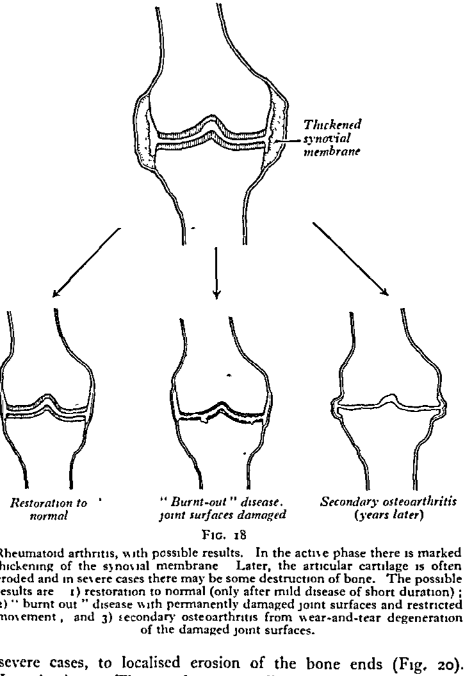

FIG. 18

Rheumatoid arthritis, with possible results. In the active phase there is marked thickening of the synovial membrane Later, the articular cartilage is often eroded and in severe cases there may be some destruction of bone. The possible results are 1) restoration to normal (only after mild disease of short duration); 2) "burnt out" disease with permanently damaged joint surfaces and restricted movement, and 3) secondary osteoarthritis from wear-and-tear degeneration of the damaged joint surfaces.

severe cases, to localised erosion of the bone ends (Fig. 20). *Investigations:* The erythrocyte sedimentation rate is raised during the active phase.

Until that information is available treatment should be begun with penicillin in large doses. *Local treatment :* The joint is rested, usually in a plaster splint. The fluid exudate, which is often purulent, is removed by aspiration (or, if necessary, by incision) At the same time a solution of the appropriate antibiotic drug is injected into the joint. Aspiration and injection of antibiotic solutions are repeated daily so long as the exudate continues to form. Rest is enforced until the infection is overcome, as shown by the subsidence of pyrexia and retrogression of the local signs. Thereafter active movements are encouraged in order to restore the greatest possible function to the joint.

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and phenylbutazone.[1] Artificial pyrexia induced by typhoid and paratyphoid antigens (TAB) is effective in certain types of case (and in Reiter's syndrome). Cortisone and adrenocorticotrophic hormone (ACTH) both produce dramatic alleviation of the symptoms, but side-effects prohibit their continued use and the symptoms return with unreduced severity when the drug is withdrawn. *Local treatment :* The treatment required for the joints themselves depends upon the activity and the severity of the inflammatory reaction. When the reaction is severe, rest or even temporary immobilisation is required. When it is moderate or slight, exercises and active use within the limits of pain are encouraged. Various physiotherapeutic measures are employed in attempts to reduce discomfort and to hasten resolution. In suitable cases a trial may certainly be given to local heat by infra-red radiation, short-wave diathermy, hot packs, or wax baths ; or to hydrotherapy.

In the later stages, operative treatment is sometimes required for increasing pain and disability, due in most cases to superimposed osteoarthritic changes. In certain joints (notably the elbow, hip, and metatarso-phalangeal joint of the great toe) arthroplasty often gives satisfactory results ; but in joints such as the wrist, knee, and ankle arthrodesis is the operation of choice.

TUBERCULOUS ARTHRITIS

In Great Britain the incidence of tuberculous arthritis has decreased in recent years, largely because of the almost universal pasteurisation of milk. In most juvenile cases seen now there is a history of contact with open pulmonary tuberculosis, often in a parent. Nevertheless bovine infection is still prevalent in Ireland, as well as in many other parts of the world.

The last few years have also seen important new developments in treatment, chief among which has been the use of streptomycin and other antibiotic drugs. Whereas formerly a joint affected with tuberculosis was inevitably destroyed, full recovery of function can now be hoped for in a reasonable proportion of cases.

Cause. Tuberculous arthritis is caused by infection of a joint with tubercle bacilli, human or bovine.

Pathology. No joint is immune, but the joints most often

[1] Trade name Butazolidin.

Diagnosis. The clue to the diagnosis is the simultaneous involvement of several joints, with raised sedimentation rate. A search should always be made for evidence of one of the distinct medical entities that may be associated with joint changes of a rheumatoid type. The most important of such conditions are: 1) Still's disease (confined to children; spleen and lymphatic glands enlarged); 2) Reiter's syndrome (urethritis, arthritis, conjunctivitis, and hyperkeratotic eruptions on the skin); 3) lupus erythematosus (scaly erythema of face or other parts); and 4) psoriasis.

FIG. 19

FIG. 20

Figure 19—Long-established rheumatoid arthritis of the knee. Note the osteoporosis and loss of cartilage space. Figure 20—Destruction of the elbow in a case of severe rheumatoid arthritis of long duration

Course. There is a tendency for rheumatoid arthritis to become quiescent after remaining active for months or years. In most cases there is permanent impairment of joint function. In certain joints—especially the knees—osteoarthritis is often superimposed upon the "burnt out" rheumatoid condition, and leads to increasingly severe disability even though the original rheumatoid affection is no longer active.

Treatment. *Constitutional treatment* is directed towards improving the general health. No specific cure has yet been found. Certain drugs appear to be beneficial in a proportion of cases, though none can be relied upon. The most useful are aspirin, gold salts,

tuberculous inflammatory reaction which is of characteristic type, with round-cell infiltration and giant-cell systems. Unless the disease is arrested, the articular cartilage is soon destroyed and the underlying bone is eroded. Sometimes the infection begins in the end of the bone, whence it extends into the joint by direct continuity. The formation of an abscess—a "cold" abscess—is a common feature. The abscess often makes its way towards the skin surface and may rupture, giving rise to a chronic tuberculous sinus. This may provide a route for the entry of secondary infecting organisms.

FIG 22

Tuberculous arthritis of the right hip. The normal side is shown for comparison. Note the osteoporosis, loss of joint cartilage, and erosion of bone surfaces.

Healing is by fibrosis. If it occurs before the articular cartilage and bone have been damaged the function of the joint is restored virtually to normal. But if cartilage or bone is damaged before healing is secured permanent impairment—often complete loss of function—is inevitable (Fig. 21).

Clinical features. Children and young adults are most commonly affected. There is often a history of contact with a patient with active pulmonary tuberculosis. In general, the predominant symptoms are pain, swelling, and impairment of function of the affected joint. *On examination* the characteristic features are increased warmth of the overlying skin, swelling from synovial

affected are the large joints such as the hip and knee, and the symphysial joints between the vertebral bodies. The organisms

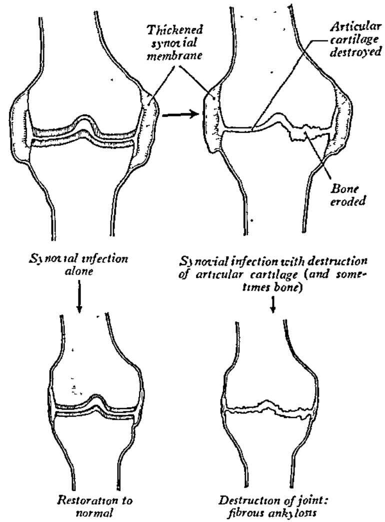

FIG. 21

Tuberculous arthritis, with possible results. When infection is purely synovial there is marked thickening of the synovial membrane but the articular cartilage is intact (note similarity to early rheumatoid arthritis) With efficient treatment begun at this stage restoration to normal is possible. More often the disease progresses to involve the articular cartilage and bone The joint is destroyed, and fibrous ankylosis is the natural outcome

reach the joint through the blood stream from a focus elsewhere. The synovial membrane is much thickened (Fig. 21) by the

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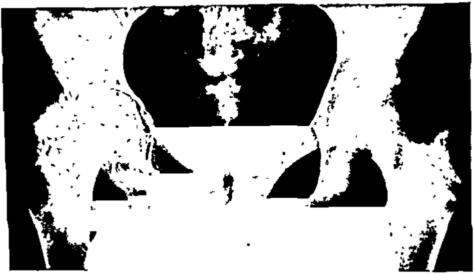

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thickening, and limitation of movement in all directions. Forced [illegible]

covered elsewhere in the body. *Radiographic examination :* The earliest change in tuberculous arthritis is diffuse osteoporosis throughout a fairly wide area of bone adjacent to the joint. If the disease is arrested early there may be no further change. But if the infection progresses the cartilage space is narrowed and the underlying bone eroded (Fig. 22) As the disease heals the bones "harden up" again—that is, the osteoporosis becomes gradually less apparent until the bone density is restored to normal. *Investigations :* The erythrocyte sedimentation rate is raised in the active stage. Its gradual decrease is an indication of healing. The Mantoux test is positive. Aspiration of the joint may yield a little [illegible]

tubercle bacilli. Biopsy of thickened synovial membrane shows the typical histological features of tuberculosis.

Complications. These are : (1) sinus formation ; (2) secondary infection through a sinus track ; (3) spread of disease to another part of the body. Other complications are peculiar to special regions—for example, compression of the spinal cord by an abscess in tuberculosis of an intervertebral joint.

Course. Under favourable conditions there is a tendency towards slow healing by fibrosis. With early treatment a movable or even a normal joint is sometimes preserved, especially in children. But in many cases the joint is largely or totally destroyed

Treatment. The principles of treatment are the same no matter which joint is affected ; variations in detail will be discussed in the sections on the individual joints. Treatment should be carried out in a country orthopaedic hospital *Constitutional treatment :* This is by rest, fresh air, adequate diet, and systemic chemotherapy. It is recommended that streptomycin, para-amino-salicylic acid (PAS), and isonicotinic acid hydrazide (INAH) be given together in a course of treatment extending over six months, provided there is no toxic reaction *Local treatment :* Initially, the joint should be rested in a splint or plaster for six months—that is, during the

Local Treat- — - Rest in splint for 6 months
- Abscess drainage

course of chemotherapy. Meanwhile abscesses should be aspirated or drained surgically. The subsequent management depends upon the state of the joint and the response to treatment, which should be assessed at the end of the first six months. If at that time the articular cartilage and bone are still intact, if the general health is good and the local signs have subsided, and if the erythrocyte sedimentation rate has shown a steady improvement, there is a good prospect that the disease has been aborted. In that event active joint movements are encouraged and gradually increased, under supervision, until adequate function is restored.

On the other hand, if the review at the end of the first six months' treatment shows that articular cartilage or bone has been destroyed, the joint must be "written off" as a functional unit, and in the case of most joints a sound bony fusion should be the ultimate objective. To this end immobilisation is continued for many months until the disease is quiescent. Thereupon arthrodesis is undertaken if necessary.

OSTEOARTHRITIS

(Hypertrophic arthritis; degenerative arthritis, osteoarthrosis; post-traumatic arthritis)

Osteoarthritis is a degenerative wear-and-tear process occurring in joints that are impaired by age, vascular insufficiency, or previous disease or injury. It is by far the commonest variety of arthritis.

Cause. It is caused by wear and tear. If a joint were never put under stress it would never become osteoarthritic. Hence the relatively lightly stressed joints of the upper limb are, in general, less prone to osteoarthritis than the heavily stressed joints of the lower limb. Nearly always, however, there is a predisposing cause that accelerates the wear-and-tear process. Almost any *abnormality of a joint may be responsible, indirectly*, for the development of osteoarthritis—often many years later. The main predisposing factors are: 1) senility—that is, an impaired capacity for tissue repair inherent in the process of aging; 2) irregularity of joint surfaces from previous fracture; 3) internal derangements, such as a loose body or a torn cartilage; 4) previous disease, leaving a damaged articular cartilage (for example, rheumatoid arthritis or haemophilia); 5) mal-alignment of a joint from any cause (for example, bow-leg); 6) obesity and overweight.

Pathology. Any joint may be affected, the lower limb joints more often than the upper. The articular cartilage is slowly worn away until eventually the underlying bone is exposed (Fig. 23). This subchondral bone becomes hard and glossy ("eburnation"). Meanwhile the bone at the margins of the joint hypertrophies to form a rim of projecting spurs known as osteophytes. There is no primary change in the capsule or synovial membrane, but the recurrent strains to which an osteoarthritic joint is subject often lead to slight thickening and fibrosis.

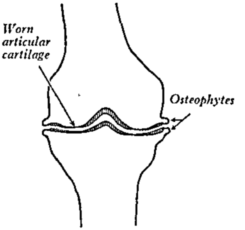


FIG. 23

Osteoarthritis The main changes are in the articular cartilage and underlying bone The cartilage is gradually worn away, disappearing first at the points of greatest pressure. The subchondral bone becomes sclerotic, and at the joint margins it hypertrophies to form osteophytes.

Clinical features. Most patients with osteoarthritis are past middle age. When it occurs in younger patients there is usually a clear predisposing cause such as previous injury or disease of the joint. The onset is very gradual, with pain that increases almost imperceptibly over months and years. Movements slowly become more and more restricted. In some joints (notably the hip) deformity is a common feature in the later stages. *On examination* slight thickening is often found on palpation, it is mainly a bony thickening caused by the marginal osteophytes There is no increased warmth. Movements are impaired slightly or markedly according to the degree of arthritis, in most joints movement is accompanied by palpable or audible crepitation of a rather coarse type. Fixed deformity (that is, inability of the joint to assume the neutral anatomical position) is often found in the hip, and sometimes in other joints. *Radiographic examination* The characteristic features of osteoarthritis are : 1) diminution of cartilage space; 2) subchondral sclerosis; and 3) spurring or "lipping" of the joint margins from the formation of osteophytes (Fig 24)

Diagnosis. This is usually made clear by the history, clinical findings, and radiographic features. Osteoarthritis is not easily confused with inflammatory forms of arthritis, for there is no synovial thickening, no increase of local skin temperature, and

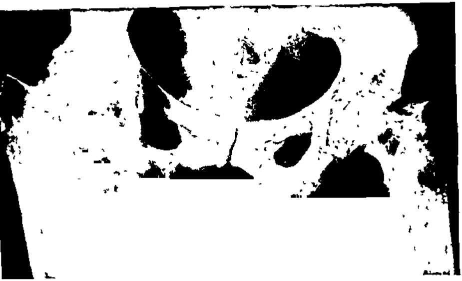


FIG. 24

Osteoarthritis of the right hip, with the sound hip shown for comparison. Note the narrowing of the cartilage space, the subchondral sclerosis, and the marginal osteophytes. There is an adduction deformity.

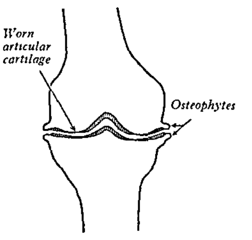
no muscle spasm ; the radiographs show sclerosis rather than osteoporosis, and the erythrocyte sedimentation rate is not increased

Course. Osteoarthritis usually increases slowly year by year. In many cases the disability never reaches the stage at which treatment is required In others increasing pain, stiffness, or deformity drives the patient to demand active measures to secure relief

Treatment. The management of osteoarthritis exemplifies well the three categories of treatment that should be considered in every orthopaedic problem—namely, 1) no treatment ; 2) conservative treatment ; and 3) operative treatment.

In many cases no treatment is required. The patient may have sought advice only because of anxiety lest some grave disease be present. Reassurance, with advice to restrict the wear and tear on the affected joint, is all that is required.

Pathology. Any joint may be affected, the lower limb joints more often than the upper. The articular cartilage is slowly worn away until eventually the underlying bone is exposed (Fig. 23). This subchondral bone becomes hard and glossy ("eburnation"). Meanwhile the bone at the margins of the joint hypertrophies to form a rim of projecting spurs known as osteophytes. There is no primary change in the capsule or synovial membrane, but the recurrent strains to which an osteoarthritic joint is subject often lead to slight thickening and fibrosis.



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Osteoarthritis The main changes are in the articular cartilage and underlying bone. The cartilage is gradually worn away, disappearing first at the points of greatest pressure. The subchondral bone becomes sclerotic, and at the joint margins it hypertrophies to form osteophytes.

Clinical features. Most patients with osteoarthritis are past middle age. When it occurs in younger patients there is usually a clear predisposing cause such as previous injury or disease of the joint. The onset is very gradual, with pain that increases almost imperceptibly over months and years. Movements slowly become more and more restricted. In some joints (notably the hip) deformity is a common feature in the later stages. *On examination* slight thickening is often found on palpation; it is mainly a bony thickening caused by the marginal osteophytes. There is no increased warmth Movements are impaired slightly or markedly according to the degree of arthritis; in most joints movement is accompanied by palpable or audible crepitation of a rather coarse type. Fixed deformity (that is, inability of the joint to assume the neutral anatomical position) is often found in the hip, and sometimes in other joints. *Radiographic examination:* The characteristic features of osteoarthritis are: 1) diminution of cartilage space; 2) subchondral sclerosis; and 3) spurring or "lipping" of the joint margins from the formation of osteophytes (Fig 24).

Gouty deposits are also common at other sites, notably in the olecranon bursa and in the cartilage of the ear. Palpable nodular deposits in the tissues are known as tophi.

Clinical features. The patient is nearly always over 40, and more often a man than a woman. The chief clinical manifestations are arthritis and bursitis. *Arthritis :* Gout affects principally the peripheral joints such as the joints of the toes, tarsus, and ankle, and the small joints of the hands. It occurs in recurrent attacks. The first attack is usually in the great toe; later attacks may affect other joints. In an acute attack the onset is sudden—often during the night. The affected joint is swollen, red, and very painful. Joint movements are greatly restricted because of the pain. The attack subsides after a few days and the joint is normal between attacks. In chronic gout several joints are affected together. They are thickened and nodular, and painful on movement. *Bursitis :* The bursa most commonly affected by gout is the olecranon bursa. It becomes distended with fluid, and there may be palpable deposits of uric-acid salts. *Other manifestations :* Deposits of uric-acid salts (tophi) are common in the ear cartilages. They may also occur at other sites. *Radiographic examination :* In acute attacks of articular gout the joints show no radiographic abnormality. In chronic gout the deposits of uric-acid salts in the bone ends show as clear-cut erosions adjacent to the joint, for the deposits are transradiant. *Investigations :* There is sometimes a mild leucocytosis. The blood uric-acid content is high immediately before an attack, but it falls after the onset of symptoms, gradually rising again as the attack subsides. Aspiration of swollen joints may yield a small quantity of clear fluid, but never pus.

Diagnosis. Acute gout has to be distinguished from other forms of arthritis of sudden onset, especially from acute pyogenic arthritis, haemophilic arthritis, and rheumatic fever. Features suggestive of gout are: a history of previous attacks, with symptom-free intervals; a raised blood uric-acid content; the presence of tophi in the ears or elsewhere; and a favourable response to treatment by colchicum. The chronic form of gout, attacking several joints simultaneously, may simulate rheumatoid arthritis.

Course. Gout usually occurs in recurrent attacks. Early attacks subside in a few days, leaving the joint clinically normal. In

When more active treatment is called for, conservative measures should usually be tried first. The methods available include physiotherapy (local heat and muscle-strengthening exercises), analgesic drugs, and supportive bandages or appliances.

When severe disability is unrelieved by conservative treatment operation may be justified. Chief among the operations available are arthroplasty (the construction of a new joint) (p. 19) and arthrodesis (elimination of the joint by fusion of the bone ends) (p. 18) Arthroplasty is applicable only to a few joints, particularly the hip, elbow, and metatarso-phalangeal joints. For most other joints arthrodesis is the operation of choice. Further details of treatment will be given in the sections on the individual joints.

GOUTY ARTHRITIS

Gout is the clinical manifestation of a disturbed purine metabolism. It is characterised by deposition of uric-acid salts—especially sodium biurate—in connective tissues such as cartilage (of joints, or of the ear), the walls of bursae, and ligaments.

Cause. The precise cause of the disturbance of metabolism is unknown. There is an inherited predisposition to the disease, and in susceptible persons attacks of gout may be induced by excessive consumption of beer or heavy wines, or by eating large amounts of purine-rich foods (liver, sweetbreads, meat). Injury to a joint may precipitate an attack.

Pathology. The primary fault is an impaired excretion of uric acid by the kidneys. In consequence the level of uric acid in the blood is increased, sometimes to as much as 6 mg. per 100 ml. (normal=3.5 mg. per 100 ml. by Folin method). In the blood the uric acid is in solution in a loose combination with proteins ; it readily comes out of solution as a sodium salt (sodium biurate) to be deposited in certain connective tissues—especially those that have been injured or those that have a sluggish blood supply, such as the articular cartilage of the joints of the foot. The deposits set up an inflammatory reaction, which is possibly allergic in nature. In acute gout the deposit is microscopic in amount and is soon reabsorbed, with restoration of the tissue to normal. In chronic gout, however, widespread deposits of sodium biurate in joint cartilages, ligaments, and the articular ends of bones, lead to considerable disorganisation of the joint.

between attacks of haemarthrosis there is moderate thickening of the joint from synovial fibrosis, movements are slightly impaired, and often there is some degree of fixed deformity.

Diagnosis. Because of the synovial thickening, increased warmth of the skin, and restriction of joint movements, haemophilic arthritis is easily mistaken for chronic inflammatory arthritis. The history of previous bleeding incidents, the sudden onset, and the recurrent nature of the attacks are important diagnostic features; and the prolonged clotting time of the blood is confirmatory evidence. Biopsy should be avoided because of the risk of post-operative bleeding.

Treatment. The principle of treatment of a recent attack is to provide rest for the joint until the haemarthrosis is absorbed. Aspiration of the blood is permissible, provided the joint is firmly bandaged and splinted afterwards. Blood transfusions are sometimes required. In the chronic degenerative phase that follows repeated haemarthroses it is often necessary to give permanent support to the joint by means of a moulded plastic splint or other appliance. Operation must be avoided.

CHARCOT'S OSTEOARTHROPATHY

(Neuropathic arthritis)

In Charcot's osteoarthropathy a joint is disorganised by repeated minor injuries because it is insensitive to pain.

Cause. The underlying cause is a neurological disorder interfering with the deep pain impulses. The commonest are tabes dorsalis (with joint complications mainly in the spine or lower limb) and syringomyelia (with joint complications mainly in the upper limb). Diabetic neuritis is an occasional cause.

Pathology. Any of the large joints may be affected, but the knee is by far the most frequent site. The elbow is the joint most often affected in the upper limb. In a normal joint harmful strains are prevented by a protective reflex whereby muscle contraction is evoked by incipient pain. When joint sensibility is destroyed the protective function of pain is lost. Strains are unrecognised and, cumulatively, they lead to severe degeneration of the joint. The changes may be regarded as a much exaggerated form of osteoarthritis The articular cartilage is worn away, but at the same time there is sometimes massive hypertrophy of bone at the

chronic gout the affected joints are gradually disorganised and permanent disability is inevitable.

Treatment. For acute attacks the most reliable remedy is colchicum given in frequent doses by mouth. Phenylbutazone is also effective The joint should be rested until the attack has subsided. Adjustments should be made to the diet in an effort to prevent or reduce further attacks. Purine-rich foods such as liver, kidneys, and sweetbreads should be avoided. Meat should be restricted. Alcohol should be avoided altogether or taken in strict moderation—especially beer and heavy wines.

HAEMOPHILIC ARTHRITIS

Although joint manifestations are common in haemophilia, examples are seen only infrequently because haemophilia is itself an uncommon disease.

Cause. Haemophilia is an inherited disease. Most types occur only in males, but are transmitted by females.

Pathology. The simplest conception (though not strictly accurate) is to regard the primary defect as an excessive stability of blood platelets, which do not break down readily enough to liberate thrombokinase. Consequently the clotting time of the blood is increased and there is a tendency to prolonged bleeding when even quite small vessels are cut or torn. Joint manifestations are caused by haemorrhage into a joint, usually after a minor strain. The joints most commonly affected are those most vulnerable to strain—especially the knee, elbow, and ankle. The joint cavity is distended with blood (haemarthrosis), which is later slowly reabsorbed if the joint is rested. Recurrent haemarthroses lead eventually to degenerative changes in the articular cartilage and to fibrosis of the synovial membrane.

Clinical features. The patient, usually a boy, is often a known sufferer from haemophilia or can recall previous bleeding incidents. He suddenly finds that a joint has become painful and swollen. *On examination* the findings vary according to the phase and duration of the arthritis For several weeks after the acute onset the joint is swollen—partly from effused blood and partly from the synovial thickening that results from interstitial extravasation. The overlying skin is abnormally warm. Joint movements are restricted and cause pain if forced. In the quiescent phase

between attacks of haemarthrosis there is moderate thickening of the joint from synovial fibrosis, movements are slightly impaired, and often there is some degree of fixed deformity.

Diagnosis. Because of the synovial thickening, increased warmth of the skin, and restriction of joint movements, haemophilic arthritis is easily mistaken for chronic inflammatory arthritis. The history of previous bleeding incidents, the sudden onset, and the recurrent nature of the attacks are important diagnostic features; and the prolonged clotting time of the blood is confirmatory evidence. Biopsy should be avoided because of the risk of post-operative bleeding.

Treatment. The principle of treatment of a recent attack is to provide rest for the joint until the haemarthrosis is absorbed. Aspiration of the blood is permissible, provided the joint is firmly bandaged and splinted afterwards. Blood transfusions are sometimes required. In the chronic degenerative phase that follows repeated haemarthroses it is often necessary to give permanent support to the joint by means of a moulded plastic splint or other appliance. Operation must be avoided.

CHARCOT'S OSTEOARTHROPATHY

(Neuropathic arthritis)

In Charcot's osteoarthropathy a joint is disorganised by repeated minor injuries because it is insensitive to pain.

Cause. The underlying cause is a neurological disorder interfering with the deep pain impulses. The commonest are tabes dorsalis (with joint complications mainly in the spine or lower limb) and syringomyelia (with joint complications mainly in the upper limb). Diabetic neuritis is an occasional cause.

Pathology. Any of the large joints may be affected, but the knee is by far the most frequent site. The elbow is the joint most often affected in the upper limb. In a normal joint harmful strains are prevented by a protective reflex whereby muscle contraction is evoked by incipient pain. When joint sensibility is destroyed the protective function of pain is lost. Strains are unrecognised and, cumulatively, they lead to severe degeneration of the joint. The changes may be regarded as a much exaggerated form of osteoarthritis. The articular cartilage is worn away, but at the same time there is sometimes massive hypertrophy of bone at the

joint margins The ligaments become lax and the joint is unstable. Indeed it is often subluxated or dislocated.

Clinical features. The patient is usually past middle age. The main symptoms are *swelling and instability of the affected joint*. Since the joint is insensitive pain is slight or absent. *On examination* the joint is thickened, mostly from irregular hypertrophy of the bone ends. The range of movement is moderately restricted, and there is marked lateral laxity. In extreme cases the joint may be dislocated. Further examination will reveal evidence of the underlying neurological disorder. *Radiographs* show severe disorganisation of the joint. The changes are basically those of osteoarthritis, but enormously exaggerated. There are loss of cartilage space and some absorption of the bone ends, often with considerable hypertrophy of bone at the joint margins (Fig. 155, p. 230, and Fig. 242, p. 341).

Treatment. In most instances the best treatment is simply to provide support for the joint by a suitable appliance. Exceptionally, operation may be undertaken to fuse the joint. The primary neurological disorder will usually demand appropriate treatment.

ARTHRITIS OF RHEUMATIC FEVER

In adolescent children and young adults arthritic manifestations are often a prominent feature of rheumatic fever (acute rheumatism).

Cause. The cause of rheumatic fever is unknown.

Pathology. Any joint may be affected. The synovial membrane is acutely inflamed, but there is no suppuration. Clear fluid is effused into the joint.

Clinical features. The patient is usually a child over 10, or a young adult. There is constitutional illness, with malaise and pyrexia A joint becomes painful and swollen, and soon afterwards other joints are likewise affected. *On examination* an affected joint is swollen, partly from contained fluid and partly from synovial thickening. The overlying skin is warmer than normal and it may be reddened. Movements are markedly restricted, and any attempt at movement causes severe pain Other features of rheumatic fever, such as carditis, should be looked for *Radiographs* of affected joints show no alteration from the normal. *Investigations :* There is a mild leucocytosis,

Diagnosis. The arthritis of rheumatic fever has to be distinguished from other forms of arthritis—especially from acute pyogenic arthritis, rheumatoid arthritis, gout, and haemophilic arthritis—and from acute osteomyelitis. Features suggestive of rheumatic fever are: onset in adolescence; affection of several joints together or in succession; severe pain with signs of acute inflammation, but without suppuration; a mild rather than a marked leucocytosis; and a rapid favourable response to salicylates.

Treatment. The patient should be under the care of a physician rather than an orthopaedic surgeon. The accepted treatment is by rest in bed and the oral administration of salicylates.

ANKYLOSING SPONDYLITIS
(Spondylitis ankylopoietica)

As the name implies, ankylosing spondylitis is primarily a disease of the spine, though in a few cases the arthritic changes involve also the proximal joints of the limbs, especially the hips. Briefly, it is a chronic inflammatory affection of the joints and ligaments of the spine, beginning in the sacro-iliac joints. It progresses slowly, the changes gradually creeping up the spinal column from below. The natural outcome is bony ankylosis of the affected joints, but the disease may be arrested at any stage short of this.

Typically, ankylosing spondylitis affects men in early adult life. After remaining active for several years it "burns itself out," always leaving some degree of permanent stiffness of the spine.

For a more complete description the reader is referred to Chapter IV (p. 165).

DISLOCATION AND SUBLUXATION OF JOINTS

A joint is dislocated or luxated when its articular surfaces are wholly displaced one from the other, so that all apposition between them is lost. A joint is subluxated when its articular surfaces are partly displaced but retain some contact one with the other.

Dislocation or subluxation may be congenital, spontaneous, traumatic, or recurrent.

CONGENITAL DISLOCATION OR SUBLUXATION

The most important representative of this group is congenital dislocation of the hip (p. 293). Congenital club foot (talipes equino-varus) (p. 378) can be regarded as a congenital subluxation of the talo-navicular joint. Congenital displacement of other joints is rare.

SPONTANEOUS (PATHOLOGICAL) DISLOCATION OR SUBLUXATION

Displacement may occur spontaneously at any joint in consequence of a structural defect or of destructive disease. It is encountered most frequently in the spine, where the stability of the intervertebral joints may be impaired by structural defects, by previous injury, or by destructive arthritis (spondylolisthesis, pp. 140 and 178). Another example is the dislocation of the hip that sometimes complicates severe tuberculous arthritis or pyogenic arthritis. Subluxation or dislocation is also a common feature of Charcot's osteoarthropathy (p. 47).

TRAUMATIC DISLOCATION OR SUBLUXATION

Injury is by far the commonest cause of dislocations. Any joint may be affected, but those most commonly dislocated are the shoulder, elbow, ankle, and interphalangeal joints These injuries are described in textbooks of fractures and joint injuries, and they will not be considered further here.

RECURRENT DISLOCATION OR SUBLUXATION

Certain joints are liable to repeated dislocation or subluxation. Usually, but not always, there has been an initial violent dislocation which leaves permanent impairment either of the ligaments or of the articular surfaces. The joints most often affected are the shoulder (p 201), the patello-femoral joint (p 351), and the ankle (p. 377)

INTERNAL DERANGEMENTS OF JOINTS

The term internal derangement implies a localised disorder which interferes mechanically with the smooth action of a joint.

Internal derangements will be considered in three groups: 1) interposition of soft tissue; 2) loose body formation; and 3) osteochondritis dissecans.

INTERPOSITION OF SOFT TISSUE IN JOINTS

The smooth action of a joint may be obstructed by a displaced mass of soft tissue within it. The soft tissue most often responsible is an intra-articular fibrocartilage, especially a semilunar cartilage in the knee (p. 342). As a rule a fibrocartilage can be displaced only when it is torn. Other soft tissues that are occasionally interposed are synovial fringes and ligamentous tags.

Clinical features. Disorders of this type are common only in the knee. The characteristic features are recurrent sudden "locking" or giving way of the joint, with later an effusion of clear fluid within it.

LOOSE BODIES IN JOINTS

Intra-articular loose bodies may be derived from bone, cartilage, or synovial membrane. They may be entirely free within the joint or they may retain a pedicle of soft tissue.

Causes. The commonest causes of loose bodies are: 1) osteochondritis dissecans (one to three loose bodies); 2) osteoarthritis, with detached osteophytes (one to ten loose bodies); 3) chip fractures of the articular ends of bones (one to three loose bodies); and 4) osteochondromatosis (fifty to five hundred loose bodies).

Pathology. *Osteochondritis dissecans* (p 52): The loose body is derived from a part of the articular surface that undergoes necrosis and separates. *Osteoarthritis:* The bodies are derived from marginal osteophytes. They often retain firm soft-tissue attachments and may cause little trouble. *Fracture of articular margin:* Fractures only occasionally cause intra-articular loose bodies. A well recognised example is a fractured medial epicondyle which may be sucked into the elbow joint while still retaining its muscle attachments. *Osteochondromatosis:* This is a rare disease of synovial membrane A large number of villous folds become pedunculated and their bulbous extremities undergo metaplasia to cartilage. Eventually they separate from their pedicles to become free mobile bodies, and many of them become ossified. The disease may affect any joint.

Clinical features. Loose bodies do not necessarily cause symptoms unless they become jammed between the joint surfaces. The characteristic feature is sudden locking of the joint, succeeded by an effusion of clear fluid within it.

Treatment. When a loose body causes trouble it should be removed.

OSTEOCHONDRITIS DISSECANS

Osteochondritis dissecans is a localised disorder of convex joint surfaces in which a segment of subchondral bone becomes avascular and, with the articular cartilage that covers it, may slowly separate from the surrounding bone to form a loose body.

Common sites of osteochondritis dissecans. The only joints commonly affected are the knee and the elbow. In the knee the site of the lesion is nearly always the medial femoral condyle, and in the elbow, the capitulum of the humerus. Occasionally the femoral head in the hip joint, and the convex surface of the talus in the ankle joint, are affected.

Cause. The precise cause is unknown Impairment of blood supply to the affected segment of bone and cartilage—possibly by thrombosis of an end-artery—has been suggested. The significance of injury is uncertain. There is probably an inborn constitutional susceptibility to the disease, for it may occur in several joints of the same patient, or in several members of a family.

Pathology. A segment of the articular surface of a bone becomes avascular (Fig 25), and a line of demarcation slowly forms between the avascular zone and the surrounding normal bone (Fig. 26). The affected segment varies in size : it often measures about half an inch to one inch in diameter and a quarter of an inch in depth. It is always on the convex joint surface. If the segment is very small it can possibly be slowly revascularised ; but in most cases it finally separates to form a loose body in the joint, still covered by its articular cartilage (Fig. 27). The resulting cavity in the articular surface of the bone is filled with fibrous tissue, but there is inevitably some irregularity of the joint surface which predisposes to the later development of osteoarthritis

Clinical features. The patient is a young adult. The early symptoms and signs are those of a mild mechanical irritation of

the joint—namely a tendency to aching after use, with recurrent effusion of clear fluid. After separation of a fragment of bone the clinical features are those of an intra-articular loose body—recurrent sudden locking of the joint accompanied by sharp pain and followed by effusion. *Radiographs* show a clearly defined

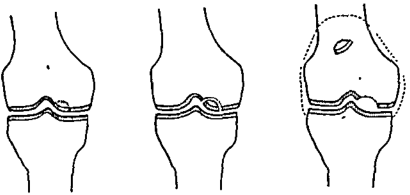

FIG. 25 FIG. 26 FIG. 27

Evolution of osteochondritis dissecans. Figure 25—Segment of articular surface deprived of blood supply. Figure 26—A line of demarcation has formed and the avascular fragment is separating from the surrounding healthy bone. Figure 27—Fragment loose in joint. A cavity remains in the articular surface.

shallow excavation into the articular surface of the bone, with an apparently separate bone fragment lying either within the cavity or elsewhere in the joint.

Treatment. Until a loose body has separated or appears " ripe " for separation treatment should be expectant. All that is necessary is to avoid excessive activity. When a fragment of bone has separated it should be removed. Further details will be found in the appropriate sections on the knee (p. 347) and the elbow (231).

OTHER MECHANICAL DERANGEMENTS OF JOINTS

Certain joints are subject to disorders of a mechanical nature, but distinct from the internal derangements already described. Examples are : 1) protrusion of an intervertebral disc ; 2) the painful arc syndrome at the shoulder ; and 3) slipping of the upper femoral epiphysis These will be described fully in the sections dealing with individual joints.

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Treatment. When a loose body causes trouble it should be removed.

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Clinical features. The patient is a young adult The early symptoms and signs are those of a mild mechanical irritation of

beneath the stripped-up periosteum, forming an investing layer known as an involucrum (Fig. 30).

Th[illegible] barrier to the spread of infecti[illegible] tly within a joint cavity [illegible] (acute pyogenic arthritis). Metaphyses that lie wholly or partly within a joint cavity include the upper metaphysis of the humerus, all the

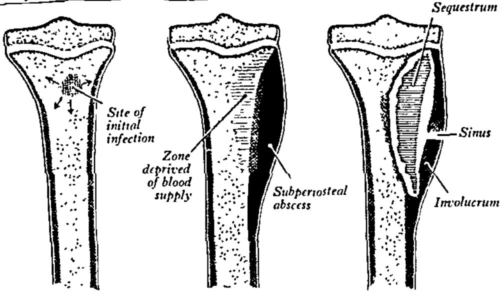


FIG 28 FIG. 29 FIG. 30

The natural evolution of a focus of osteomyelitis. Figure 28—Initial lesion in the metaphysis. Figure 29—Pus has escaped to the surface of the bone and formed a subperiosteal abscess Part of the bone has lost its blood supply from septic thrombosis of vessels Figure 30—The devitalised area eventually separates as a sequestrum. Meanwhile new bone (involucrum) is formed beneath the stripped-up periosteum ; it is perforated by sinuses through which pus escapes , This is the stage of chronic osteomyelitis. With prompt treatment the disease can often be arrested at the stage shown in Figure 28.

metaphyses at the elbow, and the upper and lower metaphyses of the femur (Fig. 31). Even when the joint is not infected it frequently swells from an effusion of clear fluid within it (sympathetic effusion).

With efficient treatment, the infection may be aborted in its earliest phase. But when it has progressed to the stage of septic thrombosis and death of bone it almost inevitably passes into a state of chronic osteomyelitis.

Osteomyelitis complicating open fracture. The organisms are introduced directly through the wound. Any part of the bone

INFECTIONS OF BONE

Infection of bone by pyogenic organisms is termed osteomyelitis.[1] It occurs in acute and chronic forms. The only other infections of bone with which the student need concern himself are tuberculous infection and syphilitic infection.

ACUTE OSTEOMYELITIS

(Acute pyogenic infection of bone)

Acute osteomyelitis is one of the important diseases of childhood ; it may also occur in adults. Early diagnosis is especially important, because the prognosis depends largely upon the interval between the onset of symptoms and the beginning of treatment.

Cause. It is caused by infection of the bone with pyogenic organisms—usually the staphylococcus, less commonly the streptococcus or pneumococcus. A minor injury to a bone renders it vulnerable to infection by organisms circulating in the blood.

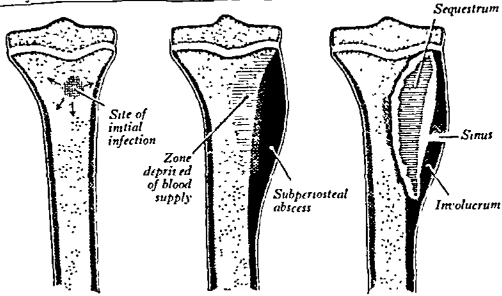
Pathology. The organisms usually reach the bone through the blood stream from a septic focus elsewhere in the body (haematogenous osteomyelitis) Less commonly they are introduced from outside, through the wound of a compound fracture. *Haematogenous osteomyelitis :* In the usual haematogenous type the infection begins in the metaphysis of a long bone (Fig. 28) ; thence it may spread to involve a large part of the bone. The organisms induce an acute inflammatory reaction, but the marshalling of the body's defensive forces is greatly handicapped in bone because its rigid structure does not allow of swelling. Pus is formed and soon finds its way to the surface of the bone where it forms a subperiosteal abscess (Fig. 29) ; later the abscess may penetrate the soft tissues and discharge at the skin surface

Often the blood supply to a part of the bone is cut off by septic thrombosis of the vessels (Fig. 29) The ischaemic bone dies and eventually separates from the surrounding living bone as a sequestrum (Fig 30). Meanwhile new bone is laid down

[1] There is nothing to be gained by distinguishing between *osteitis* (inflammation of bone) and *osteomyelitis* (inflammation of bone and bone marrow) For practical purposes the two terms may be regarded as synonymous

beneath the stripped-up periosteum, forming an investing layer known as an involucrum (Fig. 30).

The epiphysial cartilage plate is a barrier to the spread of infection, but if the affected metaphysis lies partly within a joint cavity the joint is liable to become infected (acute pyogenic arthritis). Metaphyses that lie wholly or partly within a joint cavity include the upper metaphysis of the humerus, all the



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With efficient treatment, the infection may be aborted in its earliest phase. But when it has progressed to the stage of septic thrombosis and death of bone it almost inevitably passes into a state of chronic osteomyelitis.

Osteomyelitis complicating open fracture. The organisms are introduced directly through the wound. Any part of the bone

may be affected, according to the site of injury. Suppuration and necrosis occur as in haematogenous osteomyelitis, but the pus discharges through the primary wound rather than collecting under the periosteum. The infection often becomes chronic.

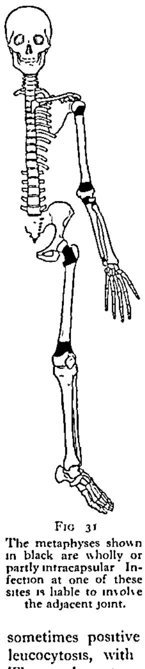

FIG 31
The metaphyses shown in black are wholly or partly intracapsular Infection at one of these sites is liable to involve the adjacent joint.

Clinical features. The haematogenous type of osteomyelitis usually occurs in children, especially boys. The bones most commonly affected are the tibia, the femur, and the humerus. The onset is rapid. The child complains of feeling ill, and of pain over the affected bone. There may be a history of recent boils or pimples. *On examination* there is obvious constitutional illness with pyrexia. Locally there is exquisite tenderness over the affected bone. The area of tenderness is clearly circumscribed; it is usually near the end of the bone in the metaphysial region. The overlying skin is warmer than normal, and often there is induration of the soft tissues; later a fluctuant abscess may be present. The neighbouring joint is often distended with clear fluid, but a good range of movement is retained (In the event of a complicating septic arthritis movement would be greatly restricted.) *Radiographic examination:* In the early stages the radiographs show no alteration from the normal (Fig. 33) Only after two or three weeks do visible changes appear, and they may never do so if efficient treatment is started very early. The important changes are diffuse osteoporosis around the metaphysial area and new bone outlining the raised periosteum (Figs. 32 and 34). *Investigations:* Blood culture is sometimes positive in the incipient stage. There is a marked leucocytosis, with preponderance of polymorphonuclear cells The erythrocyte sedimentation rate is increased.

In osteomyelitis complicating a compound fracture the temperature fails to settle after the primary treatment of the wound or rises a few days later. Pain is not a prominent feature. Re-examination of the wound reveals a purulent discharge.

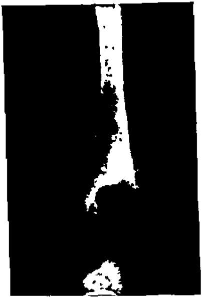

FIG. 32

Acute osteomyelitis of the femur in an infant. Radiograph three weeks after onset. Note new bone outlining the raised periosteum, and area of osteoporosis in lower metaphysis The infection spread to the knee joint

FIG. 33

FIG. 34

Acute osteomyelitis of the ulna in a child The initial film taken two days after the onset (Fig. 33) shows no abnormality Two weeks later (Fig. 34) a faint shadow along the radial side of the ulna denotes new bone formation beneath the raised periosteum.

Diagnosis. Acute osteomyelitis is to be distinguished from pyogenic arthritis of the adjacent joint by the following features: 1) the point of greatest tenderness is over the bone rather than the joint; 2) a good range of joint movement is retained; and 3) although the joint may be distended with fluid it does not contain pus (this may be confirmed by aspiration). In infants acute osteomyelitis may also be confused with scurvy (p. 93) or with syphilitic metaphysitis (p. 64).

Complications. The important complications are: 1) septicaemia or pyaemia; 2) extension of infection to the adjacent joint with consequent pyogenic arthritis (Fig. 35); and 3) retardation of growth from damage to the epiphysial cartilage (Fig. 36). Acute osteomyelitis often passes into a state of chronic infection.

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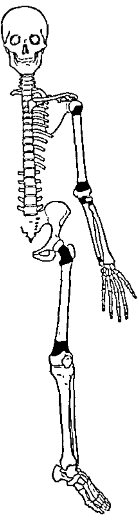

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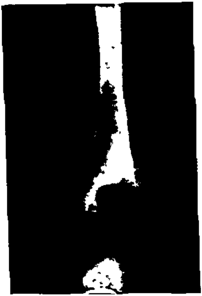

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Treatment. *Haematogenous osteomyelitis.* Efficient treatment must be begun at the earliest possible moment if there is to be a chance of aborting the infection. *General treatment:* This is by rest in bed and systemic chemotherapy. Initially, large doses of penicillin should be given, but if it is possible to isolate the causative organism and to test its sensitivity to antibiotics, one of the other drugs may be shown to be more effective for continued

FIG. 35

FIG 36

Two complications of osteomyelitis
Figure 35—Pyogenic arthritis, which led in this case to bony ankylosis.
Figure 36—Arrest of epiphysial growth, with consequent shortening.

administration. Chemotherapy should be continued for at least four weeks, even when the response has been rapid. *Local treatment:* This depends upon the stage at which the patient comes under treatment. If chemotherapy with an effective antibiotic can be begun within twenty-four hours of the onset of symptoms, and if there is no clinical evidence of an abscess, rest alone is required. But when treatment is delayed longer than twenty-four hours a subperiosteal abscess will almost certainly have formed. Operation should therefore be undertaken. An

incision is made down to the bone and subperiosteal pus is evacuated. In most cases the wound can safely be sutured. Thereafter the limb is splinted until the infection is overcome. *Osteomyelitis complicating open fracture.* The main principle of treatment is to secure free drainage through the wound, which may be enlarged if necessary for the purpose. Chemotherapy should be instituted. Later, any bone fragments that have sequestrated should be removed.

CHRONIC OSTEOMYELITIS

(Chronic pyogenic osteomyelitis)

Chronic osteomyelitis is nearly always a sequel to acute osteomyelitis. Occasionally infection is subacute or chronic from the beginning.

Cause. As with acute osteomyelitis, the staphylococcus is the usual causative organism, but streptococci, pneumococci, typhoid bacilli, or other bacteria may be responsible.

Pathology. It is commonest in the long bones. It is often confined to one end of the bone, but it may affect the whole length. The bone is thickened and generally denser than normal, though often honeycombed with areas of granulation tissue, fibrous tissue, or pus. Sequestra are commonly present within cavities in the bone. Often a sinus track leads to the skin surface: the sinus tends to heal and break down recurrently, but if a sequestrum is present it never heals permanently.

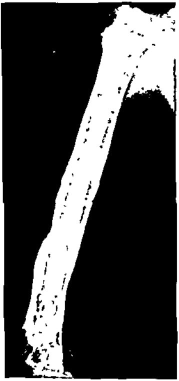


FIG. 37

Extensive chronic osteomyelitis of the humerus. Most of the original shaft has died and separated as a large sequestrum, seen lying loose within the surrounding massive involucrum

Clinical features. The main symptom is usually a purulent discharge from a sinus over the affected bone. In other cases pain is the predominant feature which brings the patient to the doctor. Discharge of pus may be

continuous or intermittent. Reappearance of a sinus that has been healed for some time is heralded by local pain, pyrexia, and the formation of an abscess. This is termed a "flare-up," or "flare," of infection. *On examination* the bone is palpably thickened, and there are nearly always a number of overlying scars or sinuses. *Radiographic examination:* The bone is thickened and shows irregular and patchy sclerosis which may give a honeycombed appearance. If a sequestrum is present it is seen as a dense loose fragment, with irregular but sharply demarcated edges, lying within a cavity in the bone (Fig. 37).

Complications. Amyloid disease may complicate long-continued chronic osteomyelitis with continuous profuse discharge of pus.

Treatment. An acute flare of chronic osteomyelitis often subsides with rest and penicillin. If an abscess forms it must be drained If there is a persistent and profuse discharge of pus a more extensive operation is advised. The aim should be to remove fragments of infected dead bone (sequestra) and to drain abscess cavities within the bone by de-roofing and "saucerising" them.

BRODIE'S ABSCESS (Chronic bone abscess)

This is a special form of chronic osteomyelitis which arises insidiously, without a preceding acute attack. There is a localised abscess within the bone, often near the site of the metaphysis. A deep "boring" pain is the predominant symptom. *Radiographically*, the lesion is seen as a circular cavity surrounded by a zone of sclerosis (Fig 38). The rest of the bone is normal. *Treatment* is by operation. The cavity is de-roofed and the pus evacuated. Whenever possible the cavity should be filled with a muscle flap to obliterate the dead space.

FIG. 38

FIG 38
Brodie's abscess The cavity in the tibia is surrounded by a zone of sclerosis

TUBERCULOUS INFECTION OF BONE

Tuberculous infection of bone is uncommon except in the vertebral bodies and in association with tuberculous infection of joints. Occasionally it occurs as an isolated lesion of a long bone or of a bone of the hand or foot.

Pathology. Tubercle bacilli reach the bone either through the blood stream or by direct extension from an adjacent focus in joint or soft tissue. There is a typical tuberculous inflammatory reaction. Part of the bone is destroyed and replaced by granulation tissue. A tuberculous abscess is commonly formed ; it tracks beneath the soft tissues or towards the surface of the body. With treatment there is a tendency to healing by fibrosis.

Tuberculosis of a vertebra. The infection typically affects the vertebral body. It may arise initially in the bone (Fig. 39) or it may spread to the vertebra from the adjacent intervertebral disc. Tuberculous vertebral bodies collapse anteriorly but retain their full depth behind, thereby becoming wedge-shaped (Fig. 40). An abscess usually tracks downwards along the vertebral column ; it may also extend backwards to the spinal canal, where it may interfere with the spinal cord.

Juxta-articular tuberculosis. The articular ends of bones are frequently eroded by tuberculosis beginning primarily in the joint. Less often there is an isolated focus of infection within the bone (Fig. 41). From such a lesion the infection tends to spread eventually to the neighbouring joint.

Bony tuberculosis in the hand or foot. The metacarpals or phalanges are the bones most commonly affected (tuberculous dactylitis). Characteristically the bone is enlarged by a fusiform swelling which at first represents thickened and raised periosteum. Later, much of the original bone is destroyed, but at the same time new bone is laid down beneath the expanded periosteum, giving the affected metacarpal or phalanx a "distended" appearance (Fig. 42) Similar changes may affect the bones of the feet, or occasionally a long bone.

Clinical features. There is usually evidence of constitutional ill health The symptoms and signs depend upon the site of the infection. In general, pain is the initial symptom ; and at most sites it is associated with obvious swelling and often with the formation of a "cold" abscess. When the bone lesion is associated

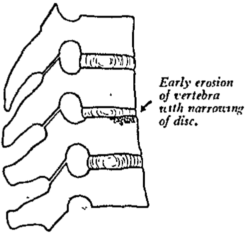


FIG. 39

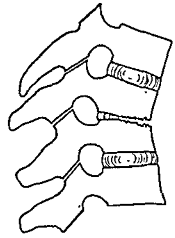


FIG. 40

Tuberculosis beginning in a vertebral body. The infection starts close to the anterior border and adjacent to an intervertebral disc (Fig. 39). It soon involves the disc and may spread to adjoining vertebrae. The bone destruction is most marked anteriorly, so the affected vertebral bodies become wedge-shaped (Fig. 40).

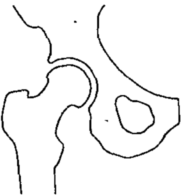


FIG. 41

Juxta-articular tuberculous focus in the neck of the femur There is no sclerosis of the surrounding bone. If unchecked by treatment, such foci eventually spread to involve the joint.

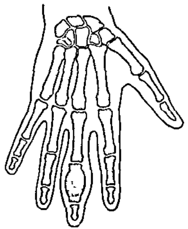


FIG. 42

Tuberculous dactylitis The affected phalanx has been "distended" by destruction of the original cortex and the laying down of new cortical bone beneath the expanded periosteum.

with tuberculous joint disease the joint symptoms predominate (see sections on the individual joints). *Radiographic examination :* The typical radiographic features of tuberculous infection of bone are: 1) diffuse osteoporosis around the site of infection; 2) erosion or "eating away" of bone, leaving a fluffy ill defined outline with no suggestion of a surrounding zone of sclerosis; and 3) in many cases a shadow in the soft tissues, denoting abscess formation. *Investigations :* The erythrocyte sedimentation rate is raised. The Mantoux test is positive. Aspirated pus is yellow and creamy: only occasionally can organisms be identified by direct examination, but culture or guinea-pig inoculation may prove its tuberculous nature. Biopsy of affected bone or surrounding soft tissue will show the histological features of tuberculosis.

Diagnosis. The diagnosis can often be presumed with fair certainty from a consideration of the history, clinical features, and radiographic findings. Features that lend support are a history of contact with tuberculosis, a strongly positive Mantoux test (in children), a raised erythrocyte sedimentation rate, and evidence of a tuberculous lesion elsewhere. The diagnosis can be proved only by identifying the causative organism or by demonstration of the typical histological features in excised fragments of tissue.

Treatment. In most instances tuberculosis of bone is associated with infection of a joint, and the treatment is mainly that of the joint lesion (tuberculous arthritis, p. 37). The treatment of an isolated tuberculous focus in bone is along similar lines. *Constitutional treatment :* This consists of rest in good surroundings, adequate diet, and systemic chemotherapy. The recommended programme of chemotherapy is to give streptomycin, para-amino-salicylic acid (PAS), and isonicotinic acid hydrazide (INAH) together in standard doses for a six months' course, provided no toxic reaction occurs. *Local treatment :* The principles are to provide prolonged rest or immobilisation for the affected part, and to remove collections of pus by aspiration or, sometimes, by operative drainage followed by immediate suture of the wound. Rest is continued until the disease becomes quiescent, as judged from improvement in the general health and weight, decrease of erythrocyte sedimentation rate, and improved radiographic appearances.

SYPHILITIC INFECTION OF BONE

In Great Britain syphilitic infection of bone is uncommon. But it is still common in some parts of the world, and it is important that the possibility of its occurrence should be borne constantly in mind. Bone changes are a late manifestation of acquired syphilis, but they may appear early in life in patients with congenital syphilis Syphilis of bone can take many forms. Only two will be described here: 1) syphilitic metaphysitis of infants; and 2) osteo-periostitis (combined osteitis and periostitis) in children or adults.

Syphilitic Metaphysitis

This is an affection of young infants with congenital syphilis. **Pathology.** Several metaphyses are affected. The zone of temporary calcification next to the epiphysial cartilage—normally seen in section as a thin grey line—is widened and yellowish. The adjacent part of the metaphysis is partly replaced by granulation tissue.

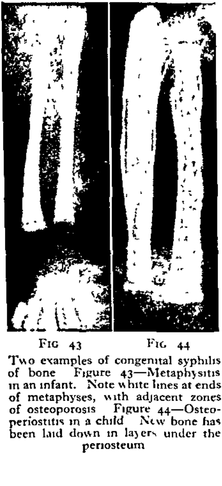


Fig 43 Fig. 44

Two examples of congenital syphilis of bone Figure 43—Metaphysitis in an infant. Note white lines at ends of metaphyses, with adjacent zones of osteoporosis Figure 44—Osteo-periostitis in a child New bone has been laid down in layers under the periosteum

Clinical features. The condition affects infants in the first six months of life. There may be severe local pain, in consequence of which the child refuses to use the affected limb (pseudo-paralysis). there is no actual loss of muscle power. *On examination* the metaphysial region is thickened and tender. *Radiographs* are characteristic The part of the metaphysis adjacent to the epiphysial cartilage shows a zone of sclerosis whereas the rest of the metaphysis is osteoporotic (Fig 43) Often there is also evidence of periostitis in the form of sub-

periosteal new bone in the metaphysis and adjacent part of the shaft. *Investigations:* The Wassermann reaction is positive.

Diagnosis. The condition may be confused with acute osteomyelitis, but the absence of leucocytosis and the positive Wassermann reaction help to distinguish the two. Metaphysitis may also be confused with scurvy, but the other features of scurvy (haemorrhage from the gums and elsewhere) are absent. Moreover, scurvy occurs in the second six months of life rather than the first.

Treatment. Intensive antisyphilitic measures are rapidly effective.

Syphilitic Osteoperiostitis

When the diaphysis or body of a bone is infected by syphilis there is usually a combination of osteitis and periostitis, although one may predominate. Osteoperiostitis often occurs with metaphysitis in infants; it may occur separately in older children with congenital syphilis (Fig. 44), or in adults with acquired syphilis.

Pathology. Of the long bones, the tibia is most commonly affected. Other bones sometimes involved are the femur, the skull, the clavicle, and the bones of the hand or foot (syphilitic dactylitis). The bone is infiltrated with syphilitic granulation tissue, which may undergo central necrosis. The extent of the lesion varies from case to case. There may be no more than a localised thickening, or there may be a diffuse infiltration of the whole of the bone (Fig. 45). The texture of the diseased bone also varies: the bone structure may be largely replaced by granulation tissue, or the formation of much new bone may lead to well marked sclerosis.

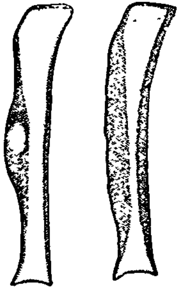


FIG. 45

Acquired syphilis of bone. Two examples of osteoperiostitis seen in diagrammatic section *Left*—Zone of destruction surrounded by localised area of thickening *Right* — Diffuse sclerotic thickening of the whole shaft.

Clinical features. Deep boring pain, worse at night, and swelling

are the predominant symptoms. *On examination* there is either a localised fusiform swelling over the shaft of the bone or a diffuse thickening of the whole length of the bone. *Radiographic examination :* Syphilitic bone disease is represented by a variety of radiographic changes ranging from severe osteoporosis to dense sclerosis. The commonest appearances are: 1) widespread subperiosteal new bone (Fig. 44); 2) a localised area of destruction or osteoporosis, with dense thickening of the overlying cortex; and 3) diffuse sclerotic thickening of the whole bone. Occasionally the predominant change is bone destruction without new bone formation. *Investigations :* The Wassermann reaction is positive.

Diagnosis. Syphilis of bone or periosteum is easily confused with a malignant bone tumour. Mistakes will be avoided if syphilis is considered as a possibility in every case of localised swelling in a limb. A positive Wassermann reaction lends support to the possibility, and the diagnosis is confirmed if there is a rapid improvement under antisyphilitic treatment. This therapeutic test often eliminates the need for biopsy in cases of limb swellings of doubtful nature

Treatment. The bone lesions usually respond well to intensive antisyphilitic measures.

TUMOURS OF BONE

Bone tumours are subdivided into benign or malignant types, and malignant tumours are further classified as primary or secondary (metastatic) It is necessary here to consider only four types of benign bone tumour and four types of malignant tumour—eight tumours in all

BENIGN TUMOURS OF BONE

The following types will be described: 1) osteoma; 2) chondroma, 3) osteochondroma, 4) osteoclastoma

OSTEOMA

This forms a smooth rounded prominence upon the surface of a long or a flat bone, or of a skull bone (Fig. 46) It may be composed of hard compact bone (ivory osteoma) or of spongy

bone (cancellous osteoma). Apart from visible or palpable swelling there are usually no symptoms.
Treatment. It may either be left alone or excised, according to the circumstances of each case.

CHONDROMA

Pathology. There are two forms of chondroma: in one the tumour grows outwards from a bone (ecchondroma); in the other it grows within a bone (enchondroma) (Fig. 47). Most *ecchondromata* arise in the hands or feet, or from flat bones such as the scapula or ilium They often reach a large size. *Enchondromata* are fairly common in the bones of the hands and feet: the affected bone is expanded by the tumour and its cortex is much thinned, so pathological fracture is common. Chondromata of the major long bones occur mainly in the distinct clinical condition known as dyschondroplasia (multiple chondromatosis or Ollier's disease) (p. 84). In this disorder, which begins in childhood, enchondromata arise in the region of the growing epiphysial cartilages of several bones. they interfere with normal growth at the epiphysial plate and consequently may lead to shortening or deformity. Very occasionally a chondroma becomes malignant.
Treatment. Many chondromata can safely be left alone. When a tumour causes trouble it should be removed.

OSTEOCHONDROMA

This is the commonest benign tumour of bone.
Pathology. It originates from the growing epiphysial cartilage plate, but as the bone grows in length the tumour gets "left behind" and thus appears to migrate along the shaft towards its centre It grows outwards from the bone like a mushroom (Fig 48). The stalk and part of the head of the tumour are of bone, but it is capped by cartilage. The tumour continues to enlarge until the cessation of skeletal growth. The ordinary osteochondroma is single; but in the distinct clinical condition known as diaphysial aclasis (multiple exostoses) (p. 83) the tumours affect several or many bones Rarely, malignant change occurs in one of the tumours.
Clinical features. The tumour is noticed as a circumscribed hard swelling near a joint. In severe examples of diaphysial aclasis there is interference with skeletal growth and the patient

BENIGN TUMOURS OF BONE

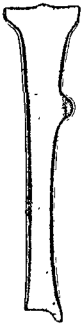


FIG 46

FIG 46
Osteoma It may occur on any bone, including those of the skull.

FIG 47
Two types of chondroma ecchondroma on proximal phalanx; enchondroma in middle phalanx (See also Figures 63 and 64)

FIG 47

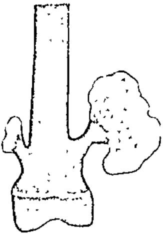


FIG 48
A small and a large osteochondroma They have originated at the growth cartilage but have "migrated" away from it with growth of the bone Each is capped by cartilage. (See also Figures 61 and 62.)

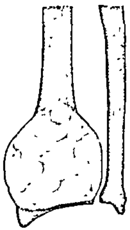


FIG 49
Osteoclastoma (giant-cell tumour) Note expansion of cortex and fine trabeculae within the tumour The tumour extends close up to the articular surface (See also Figure 50.)

may be deformed or dwarfed. *Radiographs* show the mushroom-like tumour. The stalk is often narrow.

Treatment. When necessary, the tumour should be excised.

OSTEOCLASTOMA (Giant-cell tumour)

This is an important tumour because, though generally classed as benign, it tends to recur after local removal and occasionally behaves as a frankly malignant tumour, metastasising through the blood stream. It occurs most commonly in young adults.

FIG 50

Osteoclastoma at a common site The tumour, faintly loculated, extends close up to the articular surface.

Pathology. The commonest sites are the lower end of the femur, the upper end of the tibia, the upper end of the humerus, and the lower end of the radius. Beginning in what was the metaphysial region, it extends across the site of the epiphysial cartilage into the end of the bone, often reaching almost to the joint surface (Fig 49). It destroys the bone substance, but new bone forms beneath the raised periosteum, so that the bone end becomes expanded. A few bone trabeculae remain within the tumour, giving it a loculated appearance. Pathological fracture is common

BENIGN TUMOURS OF BONE

FIG. 46

FIG 47

FIG 46
Osteoma. It may occur on any bone, including those of the skull.

FIG. 47
Two types of chondroma: ecchondroma on proximal phalanx; enchondroma in middle phalanx. (See also Figures 63 and 64.)

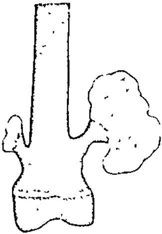


FIG 48
A small and a large osteochondroma. They have originated at the growth cartilage but have "migrated" away from it with growth of the bone Each is capped by cartilage (See also Figures 61 and 62)

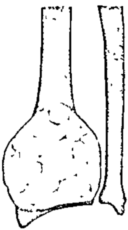


FIG 49
Osteoclastoma (giant-cell tumour) Note expansion of cortex and fine trabeculae within the tumour The tumour extends close up to the articular surface (See also Figure 50)

MALIGNANT TUMOURS OF BONE

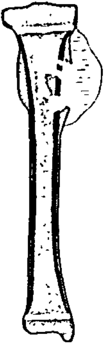


FIG 51


FIG 52

FIG. 51

Osteogenic sarcoma. It arises in the metaphysis. Note destruction of bone, raised periosteum with new bone formed beneath it, and disruption of the cortex by the tumour. The appearance is very variable.

FIG. 52

Ewing's tumour It arises in the diaphysis. Note the central area of destruction and concentric layers of subperiosteal new bone giving "onion-peel" appearance.


FIG 53


FIG 54

FIG. 53

Multiple myeloma. Small "punched out" osteolytic tumours are scattered throughout the skeleton, especially in bones containing abundant red marrow.

FIG. 54

Metastatic tumours in bone, as found in disseminated carcinoma Note the circumscribed destruction of bone without any periosteal reaction. Metastatic tumours in bone are very much more common than primary malignant bone tumours

An osteoclastoma often recurs after incomplete removal. Very occasionally it behaves as a malignant tumour and metastasises, usually to the lungs.

Clinical features. The symptoms are pain at the site of the tumour and a gradually increasing local swelling. Sometimes the patient is made suddenly aware that something is wrong by the occurrence of a pathological fracture. Examination reveals bony thickening. If the bone cortex over the tumour is thin a crepitant sensation known as "egg-shell crackling" may be felt. *Radiographs* show destruction of the bone substance, with expansion of the cortex (Fig. 50). Often the tumour extends as far as the articular end of the bone.

Treatment. This depends upon the site of the tumour. If the affected bone is one that can be reasonably dispensed with, such as the clavicle or fibula, excision of part of the bone or even the whole bone is recommended, to ensure complete removal of the tumour. But if the affected bone is one whose removal would cause severe disability, such as the femur, more conservative methods must be used. Resort should be had either to scraping out the tumour material as thoroughly as possible through a "window" cut in the cortex, to deep x-ray therapy, or to a combination of both methods. Deep x-ray therapy is capable of bringing about a permanent cure, but it is thought by some that it possibly entails a slight risk of malignant change.

MALIGNANT TUMOURS OF BONE

The following types will be described: 1) osteogenic sarcoma; 2) Ewing's tumour, 3) multiple myeloma; 4) secondary (metastatic) tumours

Osteogenic Sarcoma

This is predominantly a tumour of childhood or early adult life. When it occurs in later life it is often a complication of Paget's disease (osteitis deformans).

Pathology. The name osteogenic as used here does not imply that the tumour necessarily forms bone. It means simply that it arises from primitive bone-forming cells. The commonest sites are the lower end of the femur, the upper end of the tibia, and the upper end of the humerus The tumour begins in the

Treatment. The accepted method of treatment is by amputation. But the alternatives—local excision plus deep x-ray therapy, or deep x-ray therapy alone—should always be considered. If

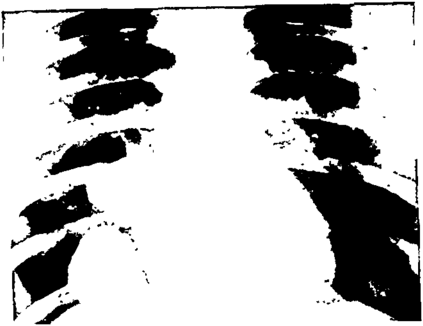


Fig. 56
Pulmonary metastases in a case of osteogenic sarcoma of the tibia.

metastases are already demonstrable it is clearly unjustifiable to subject the patient to mutilation if x-ray therapy will stem the growth even temporarily.

Ewing's Tumour (Endothelial sarcoma of bone)

Though it has a similar age incidence, occurring mainly in children and young adults, Ewing's tumour is distinct from osteogenic sarcoma in its origin, site, gross pathology, and histological appearance.

Pathology. The tumour is commonest in the shaft of the femur, tibia, or humerus; it arises in the diaphysis rather than the metaphysis of a bone. It probably develops from endothelial elements within the bone marrow. The tumour tissue is soft and vascular. As it expands it gradually destroys the bone substance. There is a striking reaction beneath the periosteum, where abundant new bone is formed in successive layers (Fig 52).

metaphysis. It destroys the bone substance and eventually bursts into the surrounding tissues, though it seldom crosses the epiphysial cartilage into the epiphysis (Fig. 51). The histological structure of the tumour varies widely: it may contain any or all of the connective tissue elements—fibrous tissue, cartilage, bone, or myxomatous tissue. (Hence the terms osteosarcoma, fibrosarcoma, and spindle-cell sarcoma used in the more precise classifications of malignant bone tumours.) The tumour metastasises early by the blood stream, especially to the lungs and to other bones.

FIG 55
Osteogenic sarcoma This is the commonest site

Clinical features. There are local pain and swelling, which gradually increase. Examination reveals a diffuse firm thickening near the end of a bone, close to the joint. The overlying skin is warmer than normal because of the vascularity of the tumour. *Radiographs* show irregular destruction of the metaphysis. Later the cortex appears to have been "burst open" at one or more places, but there are always vestiges of the original cortex. There is usually evidence of new bone formation under the corners of the raised periosteum (Codman's triangle) (Fig. 55). Occasionally well marked radiating spicules of the bone are seen ("sun-ray" appearance). A chest radiograph may show pulmonary metastases (Fig. 56)

Diagnosis. In atypical cases the condition may be confused with subacute osteomyelitis or with other bone tumours, such as osteoclastoma, Ewing's tumour, or secondary tumours. A representative piece of the tumour should be removed at biopsy for histological confirmation.

Prognosis. The mortality is in the region of 95 per cent even after amputation.

middle age. There is general ill health, with local pain at one or more of the tumour sites. *On examination* the patient is pale. There is often local tenderness over the affected bones, but there may be no obvious swelling or deformity unless pathological fracture has occurred. *Radiographs* show multiple small areas of transradiance, especially in bones containing red marrow, such as ribs, vertebral bodies, pelvic bones, skull, and proximal ends of femur and humerus (Fig. 53). Sometimes there is diffuse osteoporosis (Table II, p. 106). *Investigations:* There is microcytic anaemia. The erythrocyte sedimentation rate is increased. Bence-Jones proteose is present in the urine in more than half the cases. The serum albumen-globulin ratio is often reversed, due to a rise in the total serum globulin. Marrow biopsy usually shows typical myeloma cells.

FIG. 57
Multiple myeloma. Diffuse infiltration with numerous small tumour foci.

Diagnosis. Iliac or sternal marrow biopsy will often confirm the diagnosis when the clinical and radiographic features are equivocal

Prognosis. The tumour is uniformly fatal, though its progress can often be checked for several years.

Treatment. The tumour foci respond to deep x-ray therapy for a while. When the response is poor a trial may be made of cytotoxic poisons such as nitrogen mustard.

SECONDARY (METASTATIC) TUMOURS IN BONE

Secondary malignant tumours in bone are more common than primary tumours; but whereas most primary malignant bone tumours occur in children or young adults, secondary tumours generally occur in later life.

Pathology. The tumours that metastasise most readily to bone

Histologically the tumour consists of sheets of uniform small round cells. The tumour metastasises early through the blood stream, especially to the lungs, and sometimes to other bones.

Clinical features. Children are the usual victims. Typically, there is complaint of local pain and swelling over one of the long bones, usually about the middle of the shaft (contrast osteogenic sarcoma, which arises at the metaphysis). *On examination* the swelling is diffuse or fusiform, and of firm consistence The overlying skin is warmer than normal owing to the vascularity of the tumour. *Radiographs* show destruction of bone substance and concentrate layers of subperiosteal new bone ("onion-peel" appearance) (Fig. 52). A chest radiograph may show pulmonary metastases.

Diagnosis. In atypical cases there may be confusion with subacute osteomyelitis or with other bone tumours. Biopsy should be undertaken when the tumour is suspected.

Prognosis. The tumour is ultimately fatal, though death is sometimes averted for several years by treatment.

Treatment. The choice lies between amputation and radiotherapy. In many centres amputation is the accepted method provided there are no demonstrable metastases But it has been claimed that very high voltage deep x-ray therapy is as often effective as amputation. If this is substantiated, radiotherapy should be preferred because it obviates the psychological trauma of an amputation in a young person.

Multiple Myeloma (Plasmocytoma)

This is a uniformly fatal tumour of bone marrow, occurring in adults

Pathology. It probably arises from plasma cells It is disseminated to many parts of the skeleton through the blood stream, so that by the time the patient seeks advice the tumour foci are usually multiple, affecting chiefly the bones that contain abundant red marrow. The lesions are mostly small and circumscribed: the bone is simply replaced by tumour tissue and there is no reaction in the surrounding bone. Pathological fracture is common, especially in the spine. Histologically the tumour consists of a mass of small round cells of plasma-cell type.

Clinical features. In most cases the tumour affects adults past

middle age. There is general ill health, with local pain at one or more of the tumour sites. *On examination* the patient is pale. There is often local tenderness over the affected bones, but there may be no obvious swelling or deformity unless pathological fracture has occurred *Radiographs* show multiple small areas of transradiance, especially in bones containing red marrow, such as ribs, vertebral bodies, pelvic bones, skull, and proximal ends of femur and humerus (Fig. 53). Sometimes there is diffuse osteoporosis (Table II, p. 106). *Investigations :* There is microcytic anaemia. The erythrocyte sedimentation rate is increased. Bence-Jones proteose is present in the urine in more than half the cases. The serum albumen-globulin ratio is often reversed, due to a rise in the total serum globulin. Marrow biopsy usually shows typical myeloma cells.

FIG. 57
Multiple myeloma. Diffuse infiltration with numerous small tumour foci.

Diagnosis. Iliac or sternal marrow biopsy will often confirm the diagnosis when the clinical and radiographic features are equivocal.

Prognosis. The tumour is uniformly fatal, though its progress can often be checked for several years.

Treatment. The tumour foci respond to deep x-ray therapy for a while. When the response is poor a trial may be made of cytotoxic poisons such as nitrogen mustard.

SECONDARY (METASTATIC) TUMOURS IN BONE

Secondary malignant tumours in bone are more common than primary tumours; but whereas most primary malignant bone tumours occur in children or young adults, secondary tumours generally occur in later life.

Pathology. The tumours that metastasise most readily to bone

are carcinomas of the lung, breast, prostate, thyroid, and kidney (hypernephroma). Metastases occur most commonly in the parts of the skeleton that contain vascular marrow, especially the vertebral bodies, ribs, pelvis, and upper ends of the femur and humerus. The bone structure is simply destroyed and replaced by tumour tissue (Fig. 54). Pathological fracture is common.

FIG 58
Typical appearance of a metastatic carcinoma in the humerus, with pathological fracture The primary tumour was in the lung.

Clinical features. Pain is the predominant symptom. Sometimes the disability is insignificant until a pathological fracture occurs. The primary tumour can usually be demonstrated. *Radiographic examination :* The bone appears to have been "eaten away," so that there is a clear circumscribed area of transradiance, without any reaction in the surrounding bone (Fig. 58) Exceptionally, new bone is laid down within the metastasis, causing marked sclerosis—the exact opposite from the usual osteolytic lesion. This type is almost confined to the secondary deposits from prostatic carcinoma. In cases of diffuse infiltration there may be widespread osteoporosis (Table II, p. 106). *Investigations :* In prostatic metastases the content of acid phosphatase in the blood is increased above the normal level of 3 units per 100 ml., often to 50 or 100 units or more

Treatment. Deep x-ray therapy is a valuable palliative. Hormone therapy (stilboestrol or testosterone according to circumstances) is worth trying in metastases from the breast or prostate, and in selected cases adrenalectomy has proved well worth while in slowing the progress of the disease when other measures have failed. Local splintage may be required for pathological fracture Powerful analgesics are often necessary for relief of pain, and cordotomy is sometimes justified for the same purpose.

OTHER LOCAL AFFECTIONS OF BONE

There is a miscellaneous group of solitary lesions of bone that do not fall into the category of infection or tumour. The most important members of the group are osteochondritis, solitary bone cyst, localised fibrous dysplasia of bone, and osteoid osteoma.

OSTEOCHONDRITIS

The term osteochondritis is used to describe a group of affections of developing bony nuclei—epiphyses and apophyses—in children or adolescents. The essential features of the disease are the same no matter what part of the skeleton is affected. They are: temporary softening of the bony nucleus; liability to deformation by pressure while in the softened state; and spontaneous rehardening after an interval of about two years. It should be noted that osteochondritis is quite distinct from osteochondritis dissecans.

Common sites of osteochondritis. Osteochondritis might occur in any growing epiphysis or apophysis, but the following are those in which the affection is best recognised (Fig. 59): 1) the "ring" epiphyses of the vertebral bodies—Scheuermann's osteochondritis of the spine or adolescent kyphosis (p. 169); 2) the central epiphysis of a vertebral body—Calvé's disease (p. 171); 3) the upper epiphysis of the femur—Perthes' or Legg-Perthes' disease (p. 310); 4) the apophysis of the tibial tubercle—Osgood-Schlatter's disease (p. 356); 5) the apophysis of the calcaneum—Sever's disease (p. 393); 6) the nucleus of the navicular bone—Köhler's disease (p. 389); 7) the epiphysis of the head of the second or third metatarsal—Freiberg's disease (p. 408). Kienböck's disease of the lunate bone is in many respects similar, but it cannot be regarded as a true example of osteochondritis because it affects fully developed adult bone.

Cause. The precise cause is unknown, but the changes are believed to be due to local impairment of blood supply. There is no evidence that infection is responsible, and the significance of injury is uncertain.

Pathology. Usually the changes are confined to a single bony nucleus, but in Scheuermann's osteochondritis of the spine several vertebral bodies are affected simultaneously. The changes

are carcinomas of the lung, breast, prostate, thyroid, and kidney (hypernephroma). Metastases occur most commonly in the parts of the skeleton that contain vascular marrow, especially the vertebral bodies, ribs, pelvis, and upper ends of the femur and humerus. The bone structure is simply destroyed and replaced by tumour tissue (Fig. 54). Pathological fracture is common.

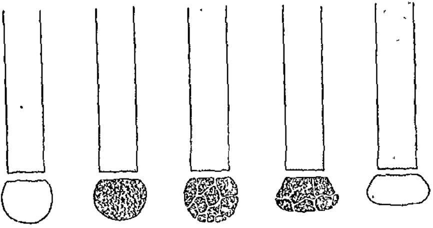
FIG. 58
Typical appearance of a metastatic carcinoma in the humerus, with pathological fracture. The primary tumour was in the lung.

Clinical features. Pain is the predominant symptom. Sometimes the disability is insignificant until a pathological fracture occurs. The primary tumour can usually be demonstrated. *Radiographic examination :* The bone appears to have been "eaten away," so that there is a clear circumscribed area of transradiance, without any reaction in the surrounding bone (Fig. 58). Exceptionally, new bone is laid down within the metastasis, causing marked sclerosis—the exact opposite from the usual osteolytic lesion. This type is almost confined to the secondary deposits from prostatic carcinoma. In cases of diffuse infiltration there may be widespread osteoporosis (Table II, p. 106) *Investigations :* In prostatic metastases the content of acid phosphatase in the blood is increased above the normal level of 3 units per 100 ml., often to 50 or 100 units or more.

Treatment. Deep x-ray therapy is a valuable palliative. Hormone therapy (stilboestrol or testosterone according to circumstances) is worth trying in metastases from the breast or prostate, and in selected cases adrenalectomy has proved well worth while in slowing the progress of the disease when other measures have failed. Local splintage may be required for pathological fracture. Powerful analgesics are often necessary for relief of pain, and cordotomy is sometimes justified for the same purpose.

Next the patchy appearance passes to one of fragmentation and the increased density is more pronounced. At this stage some flattening of the nucleus may be apparent by comparison with the normal side. Later there is a gradual return to normal bone texture, but any flattening that has occurred will remain.

Prognosis. Osteochondritis in itself is harmless, but if it leads to distortion of a joint surface it predisposes to osteoarthritis which,



1 2 3 4 5

FIG. 60

[illegible] re onset. [illegible] 3. The [illegible] softened [illegible] eformity persists The cycle occupies about two years

in the case of a large joint such as the hip, is the source of serious disability.

Treatment. In the case of osteochondritis of an apophysis which takes no part in a joint (as in Osgood-Schlatter's disease of the tibial tubercle and Sever's disease of the apophysis of the calcaneum) treatment is unimportant. Protection of the part by plaster for a month or two while the pain is severe is all that is required. The same applies to osteochondritis involving the smaller joints

In the case of osteochondritis involving a large joint—especially the hip, as in Perthes' disease—distortion of the softened epiphysis must be prevented. This can be done only by protecting the bone from the pressure of weight-bearing, preferably

go through a cycle which lasts about two years (Fig. 60). First the bony nucleus loses its normal osseous structure and becomes granular in texture. While in this state it is liable to be flattened if subjected to pressure (for instance, under body weight), and it often becomes fragmented. At this stage histological examination shows that the nucleus is avascular. Gradually, revascularisation occurs and the bone texture is restored to normal; but if deformation has been allowed to take place there is permanent alteration of shape This is important in the case of epiphyses that form part of a joint (as in Perthes' disease of the upper femoral epiphysis, in Köhler's disease of the navicular bone, and in Freiberg's disease of a metatarsal head) because the deformation of the joint surface predisposes to the later development of osteoarthritis.

Clinical features. The age at which the condition arises varies according to the particular bone affected. In general, it occurs during the stage of active development of the bony nucleus The main symptom is local pain. If the affected epiphysis forms part of a joint the function of the joint is disturbed and joint movement aggravates the pain The general health is not impaired *Radiographic examination* · The cycle of changes can be followed in serial radiographs taken at intervals of a few months. First there is a slight and often patchy increase in density of the bony nucleus

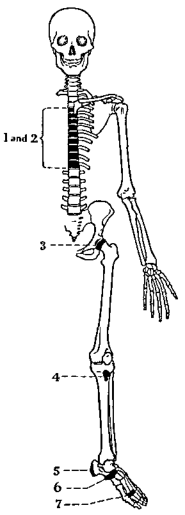


FIG. 59

FIG. 59

Common sites of osteochondritis 1 Ring epiphyses of vertebral bodies (multiple) (Scheuermann) 2 Central epiphysis of vertebral body (Calve) 3 Capital epiphysis of femur (Perthes) 4 Apophysis of tibial tubercle (Osgood-Schlatter) 5 Apophysis of calcaneum (Sever) 6 Navicular bone (Kohler) 7 Head of second or third metatarsal (Freiberg)

Next the patchy appearance passes to one of fragmentation and the increased density is more pronounced. At this stage some flattening of the nucleus may be apparent by comparison with the normal side Later there is a gradual return to normal bone texture, but any flattening that has occurred will remain.

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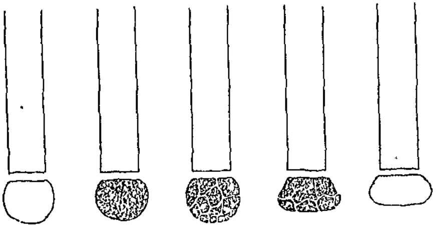

FIG 60

The cycle of changes in osteochondritis 1. Normal epiphysis before onset 2 The bony nucleus loses its normal texture and becomes granular. 3. The bone becomes fragmented 4 If subjected to pressure the softened epiphysis is flattened 5. Normal bone texture restored, but deformity persists The cycle occupies about two years.

in the case of a large joint such as the hip, is the source of serious disability.

Treatment. In the case of osteochondritis of an apophysis which takes no part in a joint (as in Osgood-Schlatter's disease of the tibial tubercle and Sever's disease of the apophysis of the calcaneum) treatment is unimportant. Protection of the part by plaster for a month or two while the pain is severe is all that is required. The same applies to osteochondritis involving the smaller joints.

In the case of osteochondritis involving a large joint—especially the hip, as in Perthes' disease—distortion of the softened epiphysis must be prevented. This can be done only by protecting the bone from the pressure of weight-bearing, preferably

by prolonged recumbency, with weight traction on the affected limb.

Further details will be found in the sections dealing with individual regions.

SOLITARY BONE CYST

(Simple bone cyst)

Solitary bone cysts occur in the long bones (especially the femur or humerus) and in the small bones of the carpus (especially the scaphoid or lunate).

Pathology. A true cyst contains fluid. It begins as a spherical lesion, but as it enlarges it tends to become oblong with its long diameter in the axis of the bone. The cyst weakens the bone and often leads to pathological fracture. Spontaneous healing has been observed, especially after a fracture has occurred through the cyst. Histologically, a bone cyst has no membranous lining. Its wall contains abundant osteoclasts.

Clinical features. Single bone cysts are encountered mostly in children or adolescents. They often cause no symptoms unless a pathological fracture occurs. *Radiographs* show a circumscribed rounded area of transradiance without a surrounding zone of sclerosis.

Diagnosis. A cyst must be differentiated from other osteolytic lesions. It may be confused with a bone abscess, with a lipoid granulomatous deposit, with localised fibrous dysplasia, or occasionally with a tumour.

Complications. The liability to pathological fracture has already been mentioned

Treatment. Small uncomplicated cysts require no treatment, but they should be kept under periodic observation. A large cyst should be curetted and packed with bone chips. After fracture, a cyst tends to heal spontaneously and operation is often unnecessary.

LOCALISED FIBROUS DYSPLASIA OF BONE

(Monostotic fibrous dysplasia)

In this condition a solitary area of bone is replaced by fibrous tissue. The cause is unknown, as also is its relationship to polyostotic fibrous dysplasia (p. 88). It is not related to the fibrous dysplasia of hyperparathyroidism.

Pathology. One of the limb bones is usually the site affected. The fibrous lesion expands at the expense of the bone, which is much weakened and may fracture.

Clinical features. There may be complaint of local pain in the affected bone *Radiographs* show a clear zone of transradiance within the bone. The area has a homogeneous " ground-glass " appearance.

Treatment. The affected segment of bone should be excised and replaced by a bone graft.

OSTEOID OSTEOMA

Osteoid osteoma is a benign circumscribed lesion of bone, of uncertain nature. It has been regarded variously as a benign tumour and as an infective lesion.

Pathology. The characteristic feature is the formation of a small nidus of osteoid tissue (seldom more than a quarter of an inch in diameter), usually in the cortex of a long bone but occasionally in cancellous bone. In the long bones the central nidus of osteoid tissue is usually surrounded by a zone of dense sclerotic bone which causes a localised thickening of the shaft. Sclerosis is slight or absent when the lesion occurs in cancellous bone.

Clinical features. The only symptom is a deep " boring " pain, worst at night. The pain is severe, but it is often eased by aspirin or its derivatives. *Radiographs* typically show local sclerotic thickening of the shaft, with a small central area of osteoporosis which, however, may be visible only in radiographs of very good quality, or in tomographs.

Diagnosis. This is mainly from osteomyelitis, and especially from Brodie's chronic bone abscess.

Treatment. The nidus of osteoid tissue should be excised together with a margin of surrounding bone.

GENERAL AFFECTIONS OF THE SKELETON

A large number of general affections of the skeleton have been described. Many of them are so rare that it is unnecessary for the student to concern himself with them. Most of the others require only brief consideration.

Classification

The conditions to be described fall into six groups (Fairbank's classification).

CONGENITAL DEVELOPMENTAL AFFECTIONS

- Osteogenesis imperfecta
- Diaphysial aclasis
- Dyschondroplasia
- Achondroplasia
- Myositis ossificans progressiva
- Multiple neurofibromatosis

ACQUIRED AFFECTIONS OF UNKNOWN ORIGIN

- Polyostotic fibrous dysplasia
- Paget's disease
- Senile osteoporosis

AFFECTIONS DUE TO ERRORS OF DIET AND METABOLISM

- Infantile scurvy
- Infantile rickets
- Other forms of rickets
- Osteomalacia
- Idiopathic steatorrhoea
- Lipoid granulomatosis
  - Gaucher's disease
  - Hand-Schüller-Christian disease

AFFECTIONS DUE TO ENDOCRINE ERRORS

- Parathyroid osteodystrophy
- Hyperpituitarism
  - Gigantism
  - Acromegaly
  - Cushing's syndrome
- Hypopituitarism
- Cretinism

AFFECTIONS DUE TO ERRORS OF THE HAEMOPOIETIC AND LYMPHATIC SYSTEMS

- Leukaemia
- Hodgkin's disease

AFFECTIONS DUE TO MULTIPLE SKELETAL TUMOURS

- Multiple myeloma
- Skeletal metastatic deposits

CONGENITAL DEVELOPMENTAL AFFECTIONS

OSTEOGENESIS IMPERFECTA

(Fragilitas ossium)

Osteogenesis imperfecta is a congenital affection in which the bones are abnormally soft and brittle. The cause is unknown. The affection is sometimes inherited.

Clinical features. In the worst cases the child is born with multiple fractures and does not survive. In the less severe examples fractures occur after birth, often from trivial violence. As many as fifty or a hundred fractures may be sustained in the first few years of life The fractures unite readily, but severe deformity often develops, either from malunion or from bending of the soft bones There is a tendency for fractures to occur less frequently in later life.

Additional features, not always present, are a deep blue coloration of the sclerotics, deafness from otosclerosis, and ligamentous laxity.

Treatment. Fractures are treated in the ordinary way. Protective appliances, such as walking calipers, may be required in older children and adults.

DIAPHYSIAL ACLASIS

(Multiple exostoses)

This is a congenital affection characterised by the formation of multiple exostoses (osteochondromata) at the metaphysial regions of the long bones. The cause is unknown. In some cases the condition is inherited.

Pathology. The fault is in the epiphysial cartilage plate. Nests of cartilage cells become displaced and give rise to bony outgrowths, which are capped by proliferating cartilage until the general cessation of skeletal growth. These exostoses, or osteochondromata, constitute one type of benign bone tumour (p 67). The number of outgrowths varies from one to hundreds; often there are between ten and twenty. In severe cases the remodelling of bone is impaired, and there may be marked deformity. Rarely, malignant change occurs in one of the tumours.

Clinical features. Usually the only symptoms and signs are those caused by the local swellings, or by their pressure effects.

In some cases there is marked deformity of the limbs. Malignant change is suggested by rapid enlargement of one of the swellings.

Fig. 61

Fig. 62

Diaphysial aclasis (multiple exostoses). Figure 61 shows stunting of the upper end of the femur due to failure of the remodelling process that accompanies normal growth. Defective remodelling is a characteristic feature of the disease in its more severe forms. Figure 62 shows typical exostoses (osteochondromata) They are always directed away from the end of the bone.

Radiographs show the bony outgrowths. In severe cases the bones are broad and ill modelled (Figs. 61 and 62).

Treatment. An outgrowth that is causing trouble should be excised

DYSCHONDROPLASIA

(Multiple chondromatosis, Ollier's disease)

In dyschondroplasia masses of unossified cartilage persist within the metaphyses of certain long bones, and the growth of the bone is retarded The condition is congenital, but the cause is unknown. Heredity plays no part.

Pathology. The fault is in the epiphysial cartilage plate In

this respect dyschondroplasia resembles diaphysial aclasis, but the two conditions are otherwise distinct. Nests of cartilage cells are displaced from the epiphysial plate into the metaphysis, where they persist as enchondromata. These masses of cartilage are regarded as a type of benign bone tumour (p. 67), although it is doubtful whether they are true neoplasms.[1]

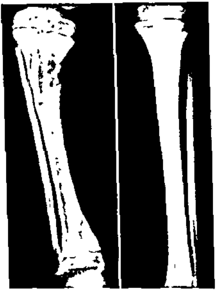

FIG 63

FIG. 64

Dyschondroplasia (multiple chondromata). Figure 63—Masses of proliferating cartilage occupy the metaphyses of one tibia. Growth is retarded and uneven. Figure 64—Multiple enchondromata in the metacarpals and phalanges.

Any bone formed in cartilage may be affected by dyschondroplasia. The more rapidly growing ends of the femur and tibia (that is, the ends near the knee) and the small long bones of the hands and feet are particularly common sites. When the major long bones are affected interference with growth at the epiphysial cartilage adjacent to the lesion may lead to serious shortening and distortion of the bone. When skeletal growth ceases the masses of cartilage ossify.

Clinical features. Limbs affected by dyschondroplasia are often short and may be markedly deformed. The hands may be grotesquely enlarged by the multiple cartilaginous swellings.

[1] It is impossible to give a precise definition of a benign tumour.

Radiographs show the multiple areas of transradiance in the affected bones (Figs. 63 and 64).

Treatment. Osteotomy may be required to correct deformities resulting from uneven growth of bone.

ACHONDROPLASIA

Achondroplasia is a congenital affection in which there is marked shortness of the limbs, with consequent dwarfing. The cause is unknown. The affection is often inherited.

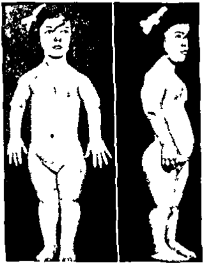

FIG. 65

Achondroplasia, showing the typical features in a child. The dwarfing is due to shortness of the limbs; development of the trunk is but little impaired

Pathology. There is a failure of normal ossification in the long bones, which may be only half their normal length. Growth of the trunk is only slightly impaired.

Clinical features. Achondroplasia is apparent at birth, the infant being strikingly dwarfed, with very short limbs that are out of proportion to the trunk (Fig. 65). Adult achondroplasiacs are seldom more than four feet in height. The hands are short and broad, the central three fingers being divergent and of almost equal length ("trident" hand). The head is slightly larger than normal, but there is no mental impairment

MYOSITIS OSSIFICANS PROGRESSIVA

Myositis ossificans progressiva[1] is a congenital affection characterised by the formation of masses of bone in the soft tissues, with consequent limitation of movement. It is often associated with shortness of the great toe or of other digits

[1] Myositis ossificans progressiva must not be confused with post-traumatic myositis ossificans The two conditions are entirely distinct. Post-traumatic "myositis ossificans" is misnamed it is nothing more than ossification within a subperiosteal haematoma.

(microdactyly). The cause is unknown. Heredity plays only a small part.

Pathology. The bone is formed by metaplasia of connective-tissue cells, not from displaced osteoblasts.

Clinical features. Changes usually appear first in early childhood. Swellings develop in the region of the neck and trunk. At first soft and perhaps transient, they later give place to hard masses of bone which lie in the course of muscles, ligaments, and fasciae Movements of the spine and ribs are reduced progressively until in the worst cases there may be total immobility. In most cases the great toe is congenitally short; the thumbs or other digits may also be short. *Radiographs* show the plates of bone in the soft tissues.

Progress. After many years increasing rigidity renders the patient bedridden. No effective treatment is known.

NEUROFIBROMATOSIS

(von Recklinghausen's disease)

This is a congenital affection characterised by pigmented spots on the skin, cutaneous fibromata, and multiple neurofibromata in the course of the cranial or peripheral nerves. The cause is unknown. Hereditary factors play a part.

Pathology. The neurofibromata consist of connective tissue arranged in whorls, with a few nerve fibres.

Clinical features. Skin lesions may be present at birth or develop later They consist of small "*café au lait*" spots, and of small fibromata which may be flat or raised. The neurofibromata may occur on any of the cranial or peripheral nerves.

The orthopaedic significance of neurofibromatosis lies mainly in the liability to scoliosis and to neurological disturbances in the limbs.

Scoliosis. Why scoliosis should occur is unknown: but it is a common complication and it sometimes progresses to an angulation so severe that the function of the spinal cord is impaired.

Neurological disturbances. These are the consequence of neurofibromata lying in the course of nerve trunks. Various limb manifestations are observed, depending upon the site of the tumour. For instance, a tumour of a nerve root within the

spinal canal may compress the spinal cord and give the typical picture of a spinal-cord tumour. Or it may compress the cauda equina or an individual nerve trunk, with consequent radiating pain and impairment of function of the involved nerve. Thus neurofibromatosis enters into the differential diagnosis of brachial pain and sciatica. *Radiographs* may show erosion of a bone where a neurofibroma lies in contact with it.

Diagnosis. The pigmented spots and cutaneous fibromata afford an important clue to the diagnosis.

Treatment. A neurofibroma that is causing trouble should be excised

ACQUIRED AFFECTIONS OF UNKNOWN ORIGIN

POLYOSTOTIC FIBROUS DYSPLASIA

Replacement of bone by fibrous tissue forms a conspicuous part of several unrelated bone diseases. In two conditions in particular, fibrous replacement is the predominant change. In one of these — parathyroid osteodystrophy — the changes are associated with hyperparathyroidism (p. 102). In the other, now to be described, there is fibrous replacement without any evidence of excessive parathyroid secretion.

Polyostotic fibrous dysplasia, then, is a condition in which parts of several bones are replaced by masses of fibrous tissue, but in which

Fig. 66

Fig 66

Polyostotic fibrous dysplasia Parts of the skeleton are replaced by fibrous tissue Unlike parathyroid osteodystrophy, this disorder is not associated with any known endocrine dysfunction.

there is no evidence of hyperparathyroidism. The cause is unknown.

Pathology. The number of bones involved varies from two or three to twelve or more. The major long bones are those mainly affected—especially the femur. The skull is also commonly involved. Affected bones are liable to bend or break.

Clinical features. The onset is in childhood but the condition is often not recognised until adult life. The main features are deformity, from bending or local enlargement of bone, and pathological fracture. The disease progresses for many years and may lead to severe crippling. *Radiographs* of the affected bones show well defined transradiant areas which often have a characteristic homogeneous or " ground-glass " appearance. The lesions are in the shaft and metaphyses rather than the epiphyses. When the lesion is extensive the cortex is expanded and thin, and the bone is bent. Sometimes the lesion has a honeycombed appearance (Fig. 66). *Investigations :* Biochemical examination of the blood shows no alteration from the normal.

The bone lesions of polyostotic fibrous dysplasia sometimes occur in association with pigmentation of the skin and, in females, sexual precocity. This combination of clinical features is known as Albright's syndrome.

PAGET'S DISEASE
(Osteitis deformans)

Paget's disease is a slowly progressive skeletal affection of one or several bones. Affected bones are thickened and spongy, and show a tendency to bend. The disease is one of the commonest general affections of the skeleton. The cause is unknown

Pathology. The bones most commonly affected are the pelvis, vertebrae, femur, tibia, and skull. Though the disease may be confined to a single bone at first, it often spreads to involve other bones later. The cortex of the bone loses its normal compact density and becomes spongy. At the same time it is widened by the formation of new bone on both its outer and inner surfaces. The whole bone is thus thickened, but the usual sharp distinction between cortex and medulla is lost. The marrow spaces are filled with fibrous tissue. In the later stages there is a tendency for the affected bones to become gradually denser,

spinal canal may compress the spinal cord and give the typical picture of a spinal-cord tumour. Or it may compress the cauda equina or an individual nerve trunk, with consequent radiating pain and impairment of function of the involved nerve. Thus neurofibromatosis enters into the differential diagnosis of brachial pain and sciatica. *Radiographs* may show erosion of a bone where a neurofibroma lies in contact with it.

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Polyostotic fibrous dysplasia, then, is a condition in which parts of several bones are replaced by masses of fibrous tissue, but in which

FIG. 66

FIG. 66

Polyostotic fibrous dysplasia. Parts of the skeleton are replaced by fibrous tissue. Unlike parathyroid osteodystrophy, this disorder is not associated with any known endocrine dysfunction.

In the spongy state the bones are softer than normal and are liable to bend. Pathological fracture may occur. In rare instances osteogenic sarcoma develops in the diseased bone.

Clinical features. The affection seldom begins before the age of 40. Often there are no symptoms, the condition being discovered incidentally during routine radiographic examination. When long bones are affected pain is sometimes complained of, but it is by no means an invariable feature. The only other symptoms arise from the bony thickening and the deformity. Thickening may be obvious clinically, especially in the case of the tibia or the skull. Thus the patient may notice that he requires progressively larger hats. Bending of the softened long bones often leads to deformity, usually in the form of anterior and lateral bowing of the femur or tibia. *Radiographic examination* (Figs. 67-69): The main radiographic features are: 1) thickening of the bone, mainly from widening of the cortex; 2) diminished density of the cortex, which loses its compact appearance and assumes a spongy or honeycombed texture; 3) marked coarsening of the bone trabeculae; 4) in the later stages a general increase of density of the affected bones. *Investigations:* The alkaline phosphatase content of the serum is increased.

Complications. The important complications are pathological fracture and, occasionally, osteogenic sarcoma.

Treatment. No treatment is necessary except when complications arise.

SENILE OSTEOPOROSIS

This condition is characterised by diffuse osteoporosis, mainly of the spine. It affects the elderly, especially women. The cause is unknown. It may possibly have an endocrine basis.

Pathology. The whole skeleton is affected to some extent, but the changes in the spine are much more marked than those elsewhere. The cortices of the vertebrae are thinner than normal, and the bone is rarefied throughout as a result of thinning of the individual trabeculae and widening of the vascular canals. Compression fracture of one or more of the vertebral bodies is liable to occur from only trivial violence. Even without fracture the thoracic vertebrae tend gradually to become wedge-shaped so that the spine bends forwards to produce a rounded kyphosis.

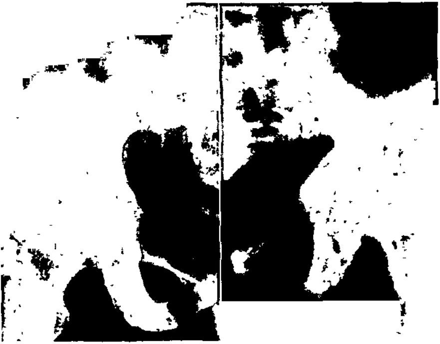

Fig. 67

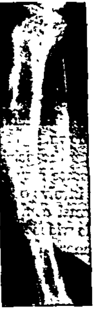

Fig 68

Fig 67

Paget's disease Half-pelvis, side by side with the normal for comparison Note the coarse trabeculae and slight distortion of the pelvic ring

Fig 68

Paget's disease Tibia, showing bending, coarsening of trabeculae, and thickened cortex which, however, is not sharply demarcated from the medulla

Fig. 69

Typical appearance of a patient with widespread Paget's disease Note the bowing of the legs, and the shortened trunk, due to collapse of softened vertebrae

Fig 69

In the spongy state the bones are softer than normal and are liable to bend. Pathological fracture may occur. In rare instances osteogenic sarcoma develops in the diseased bone.

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SENILE OSTEOPOROSIS

This condition is characterised by diffuse osteoporosis, mainly of the spine. It affects the elderly, especially women. The cause is unknown. It may possibly have an endocrine basis.

Pathology. The whole skeleton is affected to some extent, but the changes in the spine are much more marked than those elsewhere. The cortices of the vertebrae are thinner than normal, and the bone is rarefied throughout as a result of thinning of the individual trabeculae and widening of the vascular canals. Compression fracture of one or more of the vertebral bodies is liable to occur from only trivial violence. Even without fracture the thoracic vertebrae tend gradually to become wedge-shaped so that the spine bends forwards to produce a rounded kyphosis.

Clinical features. The patient is often a woman of over 60. The osteoporosis may be symptomless and may be found only by chance. In other cases there is pain in the back. The pain occurs in two forms—a mild generalised ache, and a sharper pain

FIG. 70

Senile osteoporosis Note in the photograph the rounded kyphosis and the shortened trunk. The radiograph shows marked loss of density, with thinning of the cortices. Several vertebral bodies have collapsed Differential diagnosis was from parathyroid osteodystrophy, multiple myelomatosis, diffuse carcinomatous deposits, osteomalacia, and leukaemia

of sudden onset, denoting a compression fracture. Examination reveals a rounded kyphosis in the thoracic region. If a vertebral body has collapsed there may be a more angular kyphosis with prominence of a spinous process in the thoracic or thoraco-lumbar region. The trunk is shortened and there is a transverse furrow across the abdomen (Fig 70) *Radiographic examination* The

striking feature is the reduced density of the vertebral bodies, with marked thinning of the cortices. Often there is pronounced wedging of one or more of the vertebral bodies, suggesting recent or old compression fracture (Fig. 70). The vertebra most often fractured is the first lumbar. Other parts of the skeleton may be rarefied, but in less degree. *Investigations :* The biochemistry of the blood is normal.

Diagnosis. Senile osteoporosis may be confused with other forms of diffuse osteoporosis, especially that caused by carcinomatous deposits, parathyroid osteodystrophy, Cushing's syndrome, osteomalacia, idiopathic steatorrhoea, multiple myelomatosis, or leukaemia (Table II, p. 106). The important diagnostic features, apart from the age of the patient, are the localisation of the osteoporosis predominantly in the spine, and the normal blood chemistry.

Treatment. If pain is troublesome a light spinal brace should be prescribed. Relief of symptoms sometimes follows the administration of testosterone with stilboestrol on the supposition that the condition is caused by an endocrine disturbance, but the osteoporosis itself is not influenced by treatment.

AFFECTIONS DUE TO ERRORS OF DIET AND METABOLISM

INFANTILE SCURVY

Scurvy is a haemorrhagic disease caused by a deficiency of vitamin C in the diet.

Pathology. The most striking changes are in the long bones. There is a lack of osteoblastic activity in the epiphysial growth cartilage. Haemorrhage, beginning at the epiphysial cartilage, extends beneath the periosteum, which may be raised from the bone throughout its whole length. Haemorrhages also occur from other sites, especially from the gums or within the orbit.

Clinical features. Scurvy affects infants during the second six months of life if the diet is deficient in fresh milk or other sources of vitamin C. The onset is rapid, with loss of use of a limb because of pain (pseudoparalysis). The limb is swollen and exquisitely tender over the affected bone or bones. The gums are often spongy and bleed, and there may be a " black " eye. *Radiographs* show a dense line at the junction between metaphysis

and epiphysial cartilage, with a clear band of osteoporosis on the diaphysial side (Fig. 71) Later there is ossification in the subperiosteal haematoma, as a result of which the bone is often markedly thickened (Fig. 72). *Investigations :* Ascorbic acid is absent from the plasma.


FIG 71 FIG 72

Infantile scurvy Figure 71—Early active stage Note the clear zone in the metaphysis, indicating arrest of osteoblastic activity Figure 72—Later stage, showing well marked ossification in the subperiosteal haematoma

Diagnosis. The skeletal features of scurvy are similar to those of syphilitic metaphysitis, which, however, occurs at an earlier period—namely during the first six months of life. Other distinctive features are the positive Wassermann reaction in syphilis and bleeding from the gums in scurvy. Scurvy may also be confused with acute osteomyelitis.

Treatment. The disease responds readily to the administration of vitamin C.

INFANTILE RICKETS

In rickets there is defective calcification of *growing* bone[1] in consequence of a disturbed calcium-phosphorus metabolism. With the general improvement in economic conditions infantile rickets has become rare in Britain.

Cause. Infantile rickets is caused by a deficiency of vitamin D in the diet and by inadequate exposure to sunlight, which promotes the synthesis of vitamin D in the body.

Pathology. Vitamin D promotes the absorption of calcium and phosphorus from the intestine Its deficiency therefore leads to inadequate absorption of calcium and phosphorus. The level of calcium in the blood can then be maintained only at the expense of the skeletal calcium. Proliferating osteoid tissue in the growing

[1] When similar influences act on mature adult bone the condition is known as osteomalacia

epiphyses thus remains uncalcified, and there is a general softening of the bones already formed.

Clinical features. The ordinary infantile rickets usually occurs in children about one year old. The predominant features are a large head, retarded skeletal growth, enlarged epiphyses, curvature of long bones, and deformity of the chest, which may show a transverse sulcus. In a typical case these signs produce an easily recognised clinical picture *Radiographs* show a general loss of density of the skeleton. The most striking changes, however, are in the growing epiphyses. The vertical depth of the epiphysial lines is increased, the epiphyses are widened laterally, and the ends of the shafts are hollowed out or "cupped" (Fig 73) Bending of the bones may be obvious. *Investigations :* The serum phosphorus is low. The serum calcium is normal. The alkaline phosphatase is increased.

Fig 73

Infantile rickets Note the typical widening and "cupping" of the metaphyses. The depth of the epiphysial cartilage is increased, and the general density of the bones is reduced

Diagnosis. If rickets is suspected an antero-posterior radiograph of a wrist should be obtained. The radiographic features are diagnostic of rickets, but biochemical examinations are required to indicate its type (Table I, p. 99).

Treatment. In most cases the disease responds well to vitamin D in ordinary doses In the so-called *resistant rickets* (the mechanism of which is not fully understood) massive doses of vitamin D may be needed. If severe deformities have occurred osteotomies are sometimes required for their correction.

OTHER FORMS OF RICKETS

The characteristic epiphysial changes seen in infantile rickets occur in a number of other diseases, the primary factor responsible

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The characteristic epiphysial changes seen in infantile rickets occur in a number of other diseases, the primary factor responsible

for the disordered calcium-phosphorus metabolism being different in each type. Three types will be described: coeliac rickets, renal (glomerular) rickets, and the Fanconi syndrome.

COELIAC RICKETS

Coeliac disease is a digestive disorder distinguished by an excess of fat in the stools, and frequently complicated by rachitic changes in the bones. The precise etiology is uncertain, but it has been shown that in patients with coeliac disease it is the presence in the diet of gluten (the protein fraction of flour) that is mainly responsible for the deficient absorption of fats.

The disease begins in infancy or early childhood. The general features are wasting, impaired growth, failure to gain weight, muscular hypotonia, distended abdomen, and loose offensive stools containing 40 to 80 per cent fat after drying (normal = 25 per cent). The skeletal changes, which do not develop for several years, are like those of infantile rickets. *Investigations:* The biochemical changes in the blood differ from those of infantile rickets. The serum calcium is low. The serum phosphorus is normal or low (Table I, p 99).

Treatment. There is steady improvement in the calcification of the skeleton as the primary disorder is brought under control. The diet should be free from gluten and should contain an abundant supply of calcium and vitamin D.

RENAL (GLOMERULAR) RICKETS

(Renal osteodystrophy, renal dwarfism, renal infantilism)

In renal rickets general skeletal changes are associated with chronic renal impairment The skeletal changes become manifest between the ages of 5 and 10 years The ultimate outcome is death from uraemia, usually during adolescence.

Pathology. The renal impairment may be due to congenital cystic changes, to ureteric obstruction with hydronephrosis, or to chronic nephritis The mechanism by which the renal deficiency leads to rachitic changes in the skeleton is uncertain Probably the essential factor is inadequate excretion of phosphorus by the kidneys This leads to retention of phosphorus in the blood, and its excretion in the intestine There it forms an insoluble

compound with calcium, which in consequence is not absorbed in proper amounts. The skeletal changes consist in deficient epiphysial growth and multiple deformities from bone softening. The parathyroid glands are hypertrophied, probably as a secondary effect.

Clinical features. The child is dwarfed and deformed. There are symptoms of renal impairment, such as excessive thirst and sallow complexion. The common skeletal deformities are coxa vara, genu valgum, and severe valgus deformity of the feet. *Radiographs* show epiphysial changes that are generally similar to those of infantile rickets (Fig. 74). *Investigations :* The biochemical changes are characteristic (Table I, p. 99) The serum phosphorus is markedly increased. The serum calcium is low. The blood urea is raised, often to a high figure Albumen is usually present in the urine.

FIG. 74

Rachitic changes in the epiphyses of a renal dwarf aged 14 years. There was diffuse osteoporosis of the skeleton, with multiple deformities from bending of softened bones

Treatment. This should be directed primarily against the underlying renal condition The diet should contain an abundance of calcium and vitamin D

FANCONI'S SYNDROME

(Renal tubular rickets with glycosuria)

In Fanconi's syndrome rachitic changes in the bones are associated with renal glycosuria and, usually, amino-aciduria. The primary factor is impairment of the renal tubules, which fail to reabsorb phosphorus, glucose, and certain amino acids in normal amount The excessive loss of phosphorus in the urine leads to depletion of the bone phosphate.

Clinical features. The condition begins in childhood, but at a later age than infantile rickets. There is retarded development,

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Clinical features. The condition begins in childhood, but at a later age than infantile rickets. There is retarded development,

with multiple deformities from bending of the bones, and rachitic enlargement of the epiphyses. *Radiographs* show rachitic changes at the epiphyses, generalised osteoporosis, and, in many cases, bony deformities *Investigations* (Table I, p. 99): The serum calcium is normal. The serum phosphorus is low. The alkaline phosphatase is increased The urine contains glucose.

Treatment. The intake of calcium, phosphorus, and vitamin D should be increased. Alkalies should be given to combat the associated acidosis.

RELATED DISORDERS

A number of similar disorders from renal tubular defects are recognised. In all of them there is deficient reabsorption of phosphate, but they differ in the extent to which the other functions of the tubules are impaired.

OSTEOMALACIA

Osteomalacia is the adult counterpart of infantile rickets. It is rare except in certain Asiatic countries.

Cause and pathology. As in infantile rickets, there is a deficiency of vitamin D (and often of calcium) in the diet. In consequence the intestinal absorption of calcium and phosphorus is inadequate, and calcium is withdrawn from the bones to maintain a reasonable level in the blood. The bone trabeculae are thinner than normal, and there is an abundance of poorly calcified osteoid tissue and fibrous tissue.

Clinical features. The main features are pain in the bones, and deformity Fractures are common. *Radiographs* show rarefaction of the whole skeleton The bone cortices are abnormally thin. The long bones may be curved and the pelvis triradiate. *Investigations* (Table I, p 99) The serum calcium is normal or low The serum phosphorus is low. The alkaline phosphatase is increased The calcium balance is negative.

Diagnosis. Osteomalacia must be distinguished from other causes of diffuse osteoporosis (Table II, p. 106).

Treatment. Recalcification of the skeleton is induced by adequate diet and administration of vitamin D Osteotomy may be required to correct deformity

TABLE I

SUMMARY OF THE BIOCHEMICAL CHANGES IN THE VARIOUS FORMS OF RICKETS AND OSTEOMALACIA

	Primary Fault and Mechanism	Serum Calcium.	Serum Inorganic Phosphate.	Urine	Stools.
Infantile rickets (children) Osteomalacia (adults)	Deficiency of vitamin D in diet→impaired absorption of calcium and phosphorus	Normal	Low	Normal	Normal
Cœliac rickets (children) Idiopathic steatorrhoea (adults)	Digestive deficiency→impaired absorption of vitamin D and calcium	Low	Normal	Normal	Excess of fat
Renal rickets	Impaired glomerular function→retention of phosphorus→excretion in bowel→combination with calcium preventing its normal absorption	Low	High	Albumen	Normal
Fanconi syndrome	Impaired reabsorption of phosphates by renal tubules→excessive excretion of phosphates	Normal	Low	Glucose Amino-acids Excess of phosphate	Normal

IDIOPATHIC STEATORRHOEA

This disease of adults corresponds to coeliac disease in children. It leads to skeletal changes identical with those of osteomalacia.

Cause and pathology. The cause is unknown. There is deficient absorption of fats, and consequently of the fat-soluble vitamin D and of calcium.

Clinical features. The disease may be unrecognised until a fracture occurs or a deformity becomes evident. *Radiographs*

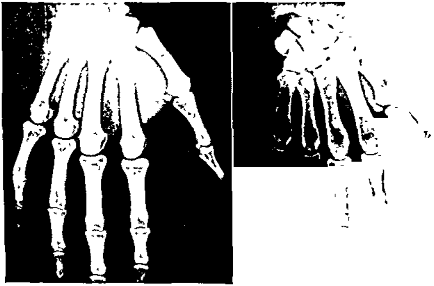

FIG. 75

Demonstration of generalised osteoporosis by radiography of one of the patient's hands side by side with a hand of a normal person of the same sex and age Note the obvious difference in bone density A case of idiopathic steatorrhoea.

show osteoporosis (Fig 75), with thinning of the cortices. *Investigations* (Table I, p. 99): The stools contain an excess of fat and of calcium The serum calcium is low. The serum phosphorus is normal The urinary excretion of calcium is low.

Diagnosis. This is from other causes of diffuse osteoporosis (Table II, p 106)

Treatment. The fat in the diet should be strictly reduced The calcium intake should be increased

LIPOID GRANULOMATOSIS

Diseases characterised by the deposition of lipoids in the cells of the reticulo-endothelial system are grouped under the general title lipoid granulomatosis. Mention will be made of two that are of interest to the orthopaedic surgeon—Gaucher's disease and Hand-Schüller-Christian disease. Both are rare.

GAUCHER'S DISEASE

In Gaucher's disease there is a deposition of the lipoid kerasin in the reticulum cells of the spleen, liver, bone marrow, and other tissues. It is believed to be due to an inborn error of metabolism. Its chief manifestations are enlargement of the spleen and cyst-like changes in the bones.

Pathology. The bone changes are due to replacement of parts of the bone by kerasin-laden reticulum cells (Gaucher cells) derived from the bone marrow.

Clinical features. The onset is in childhood but the disease may not be recognised until adolescence or adult life. The spleen is often enormously enlarged. The liver is slightly enlarged. Other features, less constant, are pain in the bones and pigmented patches in the skin and conjunctivae. The general health remains good. *Radiographs* show irregular cyst-like spaces in some of the bones, usually without enlargement of the bone. *Investigations :* Sternal puncture usually yields typical Gaucher cells.

Treatment. Splenectomy relieves local discomfort but does not cure the disease Deep x-ray therapy is sometimes effective in causing regression of the bone lesions.

HAND-SCHULLER-CHRISTIAN DISEASE

In this condition there is a proliferation of reticulo-endothelial cells, chiefly in the skull but also in other bones. Cholesterol is deposited in the lesions. The characteristic features—diabetes insipidus and exophthalmos—are explained by the localisation of the lesions in the region of the hypophysis and orbits. The cause is unknown: it probably does not represent an inborn error of metabolism like Gaucher's disease.

Pathology. The proliferations of reticulo-endothelial cells grow at the expense of the bone, producing clear-cut holes in the skull or other bones They contain characteristic " foam " cells laden with cholesterol esters

Clinical features. The symptoms arise in childhood. There may be excessive thirst with polyuria, and prominence of one or both eyes. There may be other manifestations of pituitary dysfunction, such as retarded growth. The disease progresses very slowly but is often fatal eventually. *Radiographs* show the clear-cut holes in the skull, and sometimes in other bones. There is no reaction in the surrounding normal bone.

Treatment. The lesions show a tendency to regression under treatment by deep x-ray therapy.

AFFECTIONS DUE TO ENDOCRINE ERRORS

PARATHYROID OSTEODYSTROPHY

(Hyperparathyroidism; generalised osteitis fibrosa cystica; von Recklinghausen's disease)

The characteristic features of parathyroid osteodystrophy are generalised osteoporosis of the skeleton and cystic changes in some of the bones.

Cause. It is caused by excessive parathyroid secretion, usually from an adenoma of one of the parathyroid glands.

Pathology. The excessive secretion of parathormone causes a generalised absorption of bone, the calcium from which is liberated into the blood, whence it is excreted in excessive quantities in the urine. The texture of the bone becomes spongy and the cortices are thin. Cystic changes often develop in one or more of the long bones. The kidneys frequently contain calculi.

Clinical features. The patient is adult. There are pains in the bones, indigestion, and weakness. There may also be deformity from bending of softened bone, or a pathological fracture. If the condition is untreated the patient eventually becomes bed-ridden. *Radiographs* show osteoporosis of the whole skeleton. The loss of density may be marked, and the cortices very thin. Scattered cystic changes may or may not be present in the long bones (Fig. 76). Radiographs of the renal tracts often show nephrolithiasis. *Investigations.* The changes in the blood chemistry are important in diagnosis. The serum calcium is increased but the serum inorganic phosphate is diminished. The excretion of calcium and phosphate in the urine is increased.

Diagnosis. This is from other causes of diffuse osteoporosis (Table II, p. 106).

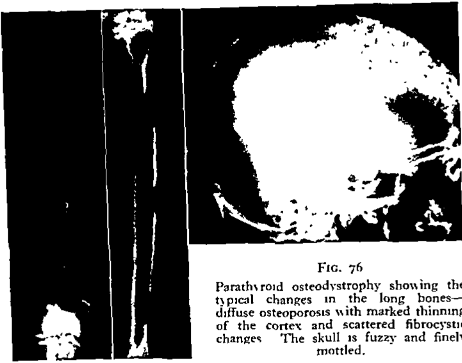

FIG. 76
Parathyroid osteodystrophy showing the typical changes in the long bones—diffuse osteoporosis with marked thinning of the cortex and scattered fibrocystic changes The skull is fuzzy and finely mottled.

Treatment. The causative parathyroid tumour should be searched for and removed.

GIGANTISM

In gigantism (one form of hyperpituitarism) skeletal overgrowth is caused by an excess of anterior pituitary hormone occurring before the growth epiphyses have fused.

Pathology. There is an eosinophilic adenoma of the anterior lobe of the pituitary. Excessive growth occurs at the epiphysial cartilages. *Despite this overactivity, epiphysial fusion occurs at* about the normal time.

Clinical features. The patient, usually a boy, may grow to a height of seven feet or more, the main increase being in the limbs. Sexual development is often impaired and the mentality may be subnormal. Signs of acromegaly often develop later.

ACROMEGALY

The primary fault is the same as in gigantism—namely, an excessive secretion of anterior pituitary hormone—but it occurs

after the epiphyses have fused. It is characterised by enlargement of the bones of the hands, feet, skull, and mandible.

Pathology. As in gigantism, there is usually an eosinophilic adenoma of the anterior lobe of the pituitary. The enlargement of the bones is caused by the deposition of new bone upon the surface of the original cortex.

Clinical features. The disease begins in early adult life. The physical features become coarse and heavy, the skin is thickened, and the hands and feet slowly enlarge. At first strong, the patient often becomes weak and sluggish later. *Radiographs* show marked enlargement of the bones of the hands and feet, and especially of the mandible. The vertebral bodies are also enlarged, especially in the antero-posterior plane.

CUSHING'S SYNDROME

This endocrine disorder is characterised by obesity, hypertrichosis, hypertension, and, in women, amenorrhoea. It may be caused either by a basophil adenoma of the pituitary or by hyperplasia or neoplasia of the adrenal cortex. Differentiation of the two types depends upon the estimation of 17-ketosteroids in the urine: in the pituitary type the 17-ketosteroid excretion is within normal limits, whereas in the adrenal type it is increased.

The orthopaedic significance of Cushing's syndrome lies in the fact that it is a cause of generalised osteoporosis of the skeleton (Table II, p. 106). The bones become soft and may be fractured by trivial violence.

HYPOPITUITARISM

Deficient secretion of the anterior lobe of the pituitary leads to various types of physical and mental underdevelopment. In the **Lorain** type the main features are marked dwarfism and sexual infantilism, often with mental backwardness In the **Fröhlich** type (dystrophia adiposo-genitalis) the predominant features are marked adiposity with impaired sexual development and sometimes with mental backwardness, but without marked dwarfism. Its importance in orthopaedic surgery arises from the fact that it predisposes to slipping of the upper femoral epiphysis (p. 312). About half the cases of slipped upper femoral epiphysis occur in patients of this type

CRETINISM

Cretinism is characterised by dwarfism with sexual and mental retardation. It is caused by congenital deficiency of thyroid secretion. From the orthopaedic viewpoint the important features are retarded growth of the limb bones and distortion of the joint surfaces. Marked improvement follows treatment with thyroid extract, but development is never entirely normal.

AFFECTIONS DUE TO ERRORS OF THE HAEMOPOIETIC AND LYMPHATIC SYSTEMS

LEUKAEMIA

Skeletal changes are a fairly common feature both of lymphatic and of myeloid leukaemia. They are probably commonest in the subacute lymphatic leukaemia of young children. The changes are due to infiltration of the bones by the proliferating white cells. The femur, humerus, spine, and pelvis are the bones most commonly affected; the changes are never distributed uniformly throughout the whole skeleton. Symptoms in the bones are seldom complained of. *Radiographic examination:* The usual changes consist of zones of osteoporosis, with delicate subperiosteal new bone formation, in the metaphysial regions of affected long bones Occasionally there is a more diffuse osteoporosis—for example, in the spine and proximal parts of the limb bones.

Diagnosis. The constitutional symptoms, with enlargement of the lymphatic glands and spleen, suggest the diagnosis, which is confirmed by blood counts and marrow biopsy (sternal puncture). If the infiltration is widespread leukaemia may be confused with other causes of diffuse osteoporosis (Table II, p 106).

HODGKIN'S DISEASE
(Lymphadenoma)

Involvement of bones in Hodgkin's disease is due to deposits of lymphadenomatous tissue. The spine, pelvis, and proximal limb bones are those most often affected. *Radiographic examination.* The bone lesions are usually osteolytic, occasionally sclerotic.

Diagnosis. This is by biopsy of an enlarged lymphatic gland.

AFFECTIONS DUE TO MULTIPLE SKELETAL TUMOURS

MULTIPLE MYELOMA

This is a cause of multiple osteolytic lesions or of diffuse osteoporosis. Its features were described in the section on bone tumours (p. 74).

MULTIPLE METASTATIC DEPOSITS

Skeletal metastases are frequently multiple, and occasionally there is a diffuse infiltration of many bones. The primary tumour is usually a carcinoma, occasionally a sarcoma. The essential features of skeletal metastases were described in the section on bone tumours (p. 75).

TABLE II

TEN CAUSES OF DIFFUSE OSTEOPOROSIS

Cause	Diagnostic Features
Prolonged recumbency	History of confinement to bed for months or years
Senile osteoporosis	Spine predominantly affected. No biochemical change in blood
Parathyroid osteodystrophy	Diagnostic biochemical changes in blood—serum calcium increased ; serum phosphate decreased.
Cushing's syndrome	Characteristic clinical features—obesity, hypertrichosis, hypertension, amenorrhoea in women
Rickets (all types)	Rachitic changes at growing epiphyses Biochemical changes depend on type of rickets (see Table I, p 99).
Osteomalacia	Dietary deficiency apparent. Characteristic biochemical changes in blood — serum calcium normal (or decreased); serum phosphate decreased
Idiopathic steatorrhoea	Excess of fat in faeces Blood changes—serum calcium decreased, serum phosphate normal
Multiple myelomatosis	Usually multiple circumscribed lesions, but may be diffuse Bence-Jones proteose often present in urine Marrow biopsy shows excess of plasma cells
Diffuse carcinomatosis	Primary tumour demonstrable
Leukaemia	Blood examination and marrow biopsy show excess of immature white cells.

INFLAMMATORY LESIONS OF SOFT TISSUE

BURSITIS

Inflammation may occur in a normally situated bursa or in an adventitious bursa. It may arise from mechanical irritation or from bacterial infection.

Irritative Bursitis

This is caused by excessive pressure or friction, occasionally by a gouty deposit. There is a mild inflammatory reaction in the wall of the bursa, and there is usually an effusion of clear fluid within the sac. Examples are the common bunion that forms over a prominent metatarsal head, infrapatellar bursitis or "housemaid's knee," olecranon bursitis (sometimes caused by gout), and subacromial bursitis.

Treatment. In many cases the inflammation subsides with rest if continued pressure or friction is prevented. In resistant cases cure can be effected only by operative excision of the bursa.

Infective Bursitis

There may be acute inflammation from infection with an organism of the pyogenic group, or chronic inflammation as in tuberculous bursitis. Examples of acute pyogenic bursitis are septic bunion and septic infrapatellar bursitis. A bursa that is sometimes affected by tuberculosis is the trochanteric bursa.

Treatment. Treatment of acute suppurative bursitis is by surgical drainage and antibiotic drugs. In chronic bursitis excision of the bursa is required.

TENOSYNOVITIS

The term tenosynovitis implies inflammation of the thin synovial lining of a tendon sheath as distinct from its outer fibrous sheath. Like bursitis, tenosynovitis may be caused by mechanical irritation or by bacterial infection.

Irritative (Frictional) Tenosynovitis

This is caused by excessive friction from over-use. The synovial sheath is mildly inflamed and there is an exudate of watery fluid within it. The commonest sites are the tendon

sheaths of the extensor pollicis longus and extensor indicis at the back of the lower forearm (p. 249).

INFECTIVE TENOSYNOVITIS

Bacterial infection of a tendon sheath may be acute or chronic. Acute infective (suppurative) tenosynovitis is caused by an organism of the pyogenic group. There is an acute inflammatory reaction in the wall of the sheath, with a purulent exudate from it. The commonest site is one of the flexor tendon sheaths in the hand (p 264). In chronic bacterial tenosynovitis the infection is usually tuberculous The synovial wall is much thickened and there is a fibrinous exudate. The flexor sheaths of the forearm and hand are the commonest sites (compound palmar ganglion, p. 264)

TENOVAGINITIS

In tenovaginitis there is a mild chronic inflammation or thickening of the fibrous wall of a tendon sheath. The cause is unknown : it is not due to bacterial infection. The only common sites are the mouths of the fibrous flexor sheaths in the fingers or thumb (" trigger " finger, p. 276), and in the sheaths of the extensor pollicis brevis and abductor pollicis longus at the radial side of the wrist (de Quervain's syndrome, p. 275).

FIBROSITIS

Fibrositis is a clinical rather than a pathological entity. Some deny its existence Certainly its nature is obscure. Nevertheless the term is a useful label for a clinical condition that at present lacks a complete explanation The main features are pain in certain muscles, with tenderness when they are gripped or squeezed. Joint movements are full and there are no other objective signs. The condition is commonest in the muscles of the back, especially in the trapezius area (p 144)

TUMOURS OF SOFT TISSUE

The soft-tissue tumours that are met with in orthopaedic practice arise from the connective tissues or blood-vessels of the limbs or trunk. They may be benign or malignant

BENIGN TUMOURS OF SOFT TISSUE

In the soft tissues of the limbs and trunk benign tumours are much more common than malignant. Four types will be described: 1) neurofibroma; 2) fibroma; 3) lipoma; 4) haemangioma.

Neurofibroma

A neurofibroma forms a rather soft, circumscribed, rounded and slightly tender swelling in the skin or deeper tissues. It arises from the interstitial tissue of a peripheral nerve. Histologically it is composed of cellular fibrous tissue arranged in whorls. The tumour may be solitary; but in the condition known as multiple neurofibromatosis (von Recklinghausen's disease) (p. 87) numerous tumours are associated with pigmented spots on the skin.

Fibroma

This is rather uncommon in the extremities. It is a firm, rounded, painless nodule, usually connected with a fascial aponeurotic structure such as a digital flexor sheath.

Lipoma

A common tumour that may arise in almost any part of the body, a lipoma forms a soft, semi-fluctuant, lobulated mass enclosed within a thin capsule. It consists of fat, usually with little connective-tissue stroma. Less often it contains an abundance of fibrous tissue, which gives it a firm consistence (fibrolipoma)

Haemangioma

Haemangioma is a benign tumour of blood vessels. A **capillary haemangioma** forms a dark red, irregular, slightly raised blotch on the skin It is usually congenital. A **cavernous haemangioma** is composed of widely dilated vascular channels with intervening connective tissue It forms a localised or diffuse tumour within the skin, subcutaneous tissue or muscle. A characteristic feature of diagnostic importance is that the tumour is compressible.

Treatment. Superficial capillary haemangiomata fall within the province of the plastic surgeon. A localised deep cavernous

haemangioma is sometimes amenable to excision. If excision is *impracticable, injection of the dilated vessels with boiling water* is sometimes effective. Extensive diffuse tumours cannot be eradicated; amputation may be required.

GIANT-CELL TUMOUR OF TENDON SHEATH

This tumour occurs almost exclusively in the hand. It is described on page 267.

MALIGNANT TUMOURS OF SOFT TISSUE

Malignant tumours of soft tissue are uncommon. Of mesenchymal origin, they arise from connective tissues such as fascia, aponeurosis, tendon sheath, intermuscular septa, voluntary muscle, and synovial membrane. The following varieties will be considered: 1) fibrosarcoma, 2) synovial sarcoma (malignant synovioma); 3) liposarcoma; 4) rhabdomyosarcoma.

FIBROSARCOMA

A fibrosarcoma is the commonest of the malignant connective tissue tumours Derived from fibroblasts, it may arise in any fibrous connective tissue

Pathology. The tumour consists of a firm rounded mass of pale pinkish tissue. It appears to be encapsulated, but the capsule forms no barrier to the spread of the tumour. Histologically it is composed of spindle cells with elongated nuclei. Metastases occur chiefly in the lungs.

Clinical features. The tumour occurs at any age. The complaint is of a progressively enlarging painless swelling. *On examination the tumour is of firm consistence.* A deep tumour is often attached to the underlying bone Apart from the swelling, there is little interference with function until the late stages.

Course. Many fibrosarcomata grow slowly and are of relatively low malignancy The prognosis is hopeful if efficient treatment is carried out at an early stage

Treatment. Local excision, with a wide margin of healthy tissue, should be undertaken whenever practicable Operation should be preceded and followed by deep x-ray therapy If local excision is impracticable, amputation will be required. In inoperable cases radiotherapy is a useful palliative measure

SYNOVIAL SARCOMA (Malignant synovioma)

This is a rare tumour which arises from the synovial lining of a joint, tendon sheath, or bursa. It is usually highly malignant.

Pathology. The tumour grows as a solid, whitish, fleshy mass which tends to follow the plane of least resistance. It invades the surrounding soft tissues and, when arising from a joint, it may invade the bone but seldom the articular cartilage. Histologically, the tumour is composed of masses of fusiform cells, but the picture is characterised by the formation of spaces or clefts lined by cuboidal cells, suggesting a synovial cavity. Metastasis occurs early through the blood stream, chiefly to the lungs.

Clinical features. Synovial sarcoma may occur at any age. The main symptoms are pain and swelling in the region of the affected joint, bursa, or tendon sheath. They develop insidiously and increase relentlessly without remission. When the swelling is superficial the overlying skin is warmer than normal. The movement of an affected joint is generally impaired only slightly, if at all. *Radiographic examination :* Usually there is no alteration from the normal. Bone may be invaded and destroyed by a tumour arising at a joint, but the cartilage space retains its normal width. Radiographs of the chest may show pulmonary metastases. *Investigations :* Biopsy reveals the characteristic histological appearance.

Diagnosis. A synovial sarcoma arising in a joint may be confused with chronic inflammatory arthritis, which may be associated with considerable thickening of the synovial membrane. A progressively increasing synovial swelling, with relatively little impairment of movement and normal radiographic findings, should always arouse suspicion of a synovial sarcoma. Biopsy should then be undertaken without delay.

Prognosis. The outlook is much worse than that for fibrosarcoma. The tumour is nearly always fatal from pulmonary metastases

Treatment. If the anatomical relations of the tumour allow it, wide local excision, with pre-operative and post-operative x-ray therapy, is recommended. When the tumour involves a joint amputation offers the best hope of cure. But if pulmonary metastases are already demonstrable amputation should usually be avoided and reliance placed upon palliative x-ray therapy.

LIPOSARCOMA

This is a rare tumour whose characteristic histological feature is the presence of foamy embryonal cells containing intracellular fat. It grows as a multilobulated mass, usually in the buttock or thigh, and it often attains an enormous size. Metastases occur mainly in the lungs.

RHABDOMYOSARCOMA

This rare variety of soft-tissue sarcoma arises in skeletal muscle. It is characterised by cells with longitudinal and cross striation, or by primitive myoblasts. Highly malignant, it grows rapidly and metastasises early, mainly to the lungs.

NEUROLOGICAL DISORDERS

POLIOMYELITIS

(Infantile paralysis)

In Great Britain the incidence of poliomyelitis has increased so much in recent years that its management—or rather the management of the paralytic disabilities that it produces—is among the foremost of present-day orthopaedic problems. It is a virus infection of nerve cells in the anterior grey matter of the spinal cord, leading in many cases to temporary or permanent paralysis of the muscles that they activate

Cause. It is caused by infection with an ultra-microscopic filterable virus, of which at least three types have been identified.

Pathology. The route of infection is uncertain Probably the virus can gain access either through the nasopharynx or through the gastro-intestinal tract. Once in the body it finds its way to anterior horn cells of the spinal cord (Fig. 77) and sometimes to nerve cells in the brain stem. According to the virulence of the infection the cells may escape serious harm, or they may be damaged or killed. If cells are damaged there is paralysis of the corresponding muscles but recovery is possible ; if the cells are killed paralysis is permanent. The extent and distribution of the lesions vary widely from case to case

Clinical features. Although it is still commonest in children, poliomyelitis often attacks young adults. For descriptive purposes the disease is conveniently divided into five stages.

Stage of incubation. This is the interval between infection and onset of symptoms. It lasts about two weeks. There are no symptoms.
Stage of onset. This lasts about two days. The symptoms are like those of influenza: headache, pains in the back and limbs, and general malaise. *On examination* there is mild pyrexia, often

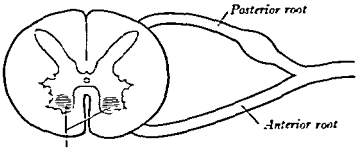

FIG. 77

Section of spinal cord. The virus of poliomyelitis attacks the anterior horn cells. If the cells are killed there is permanent paralysis of the corresponding muscle fibres. If the cells are damaged but not killed the paralysis is recoverable.

with neck rigidity if flexion is attempted, and tenderness of muscles. *Investigations:* Lumbar puncture usually reveals an increase of cells in the cerebrospinal fluid.

In many cases the disease does not progress beyond this stage, the patient making a rapid and complete recovery.
Stage of greatest paralysis. This stage, when it occurs, lasts about two months. Paralysis develops rapidly and is usually at its greatest within a few hours, thereafter remaining unchanged throughout this stage. The extent and distribution of the paralysis vary enormously. There may be no paralysis at all, or it may be total. In this stage muscle pain continues, and unparalysed muscles are often painful if stretched. If the respiratory muscles are paralysed preservation of life will be dependent upon artificial respiration.
Stage of recovery When any recovery of power occurs it may continue for about two years.[1] The degree of recovery varies

[1] It will be observed that the figure 2 appears in the stated duration of each of the first four stages—2 weeks, 2 days, 2 months, 2 years. These are only very approximate figures, but they are easily memorised.

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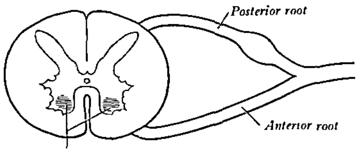

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within the widest limits. There may be complete recovery or there may be none.

Stage of residual paralysis. Paralysis or weakness persisting after two years is permanent. Its degree and extent vary from insignificant local weakness to almost total paralysis of the trunk and all four limbs.

Prognosis. In round figures, it may be stated that half of all patients clinically infected with poliomyelitis have no paralysis at any time. Of those with paralysis, 10 per cent die (usually from respiratory paralysis); 30 per cent recover fully; 30 per cent have moderate permanent paralysis; and 30 per cent have severe permanent paralysis.

Prophylaxis. Some success has been achieved in preventing or attenuating the disease by the use of a vaccine of killed virus, the injection of which stimulates the formation of antibodies. But a vaccine that is safe and fully effective has not yet been developed.

Treatment. No specific treatment is available. In few diseases is the doctor so powerless to influence recovery: the patient either will or will not recover his muscle power, depending upon the severity of the neurological damage; and there is very little that the doctor can do about it. The main duties of the orthopaedic surgeon are to prevent deformity, to assist returning muscle power by graduated exercises, and to reduce residual disability by the provision of appropriate appliances or by operations on joints or muscles. The treatment appropriate to each stage of the disease is best considered separately.

Stage of onset. The patient should rest in bed and may be given sedatives as required.

Stage of greatest paralysis. In this stage artificial respiration in a mechanical respirator ("iron lung") may be necessary to preserve life if the respiratory muscles are paralysed. Paralysed limbs may have to be supported by splints in a neutral position to prevent the development of contractures with consequent deformity. Fixed equinus deformity of the ankle and foot is particularly liable to develop in cases of paralysis of the anterior leg muscles unless the foot is maintained at a right angle to the leg. Joints should be put through a full range of movement daily, so far as pain allows. Muscle pain may be eased by warmth, as from hot packs. Whether or not the patient must remain in

bed in this stage will depend upon the degree and distribution of the paralysis.

Stage of recovery. The patient should be under the close supervision of a skilled physiotherapist. Any muscle that is seen to be

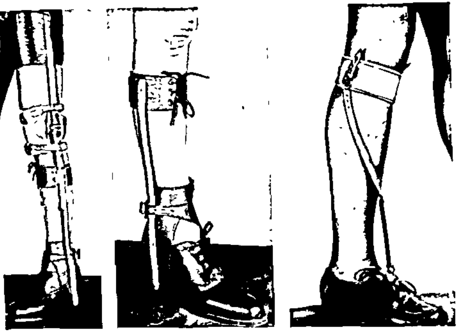

FIG. 78 FIG. 79 FIG. 80

Three types of external supportive appliance for the lower limb. Figure 78—Caliper to support the knee in quadriceps paralysis. Figure 79—Double below-knee steels for stabilising a weak ankle and foot. Figure 80—Drop-foot spring used to maintain dorsiflexion of the foot in paralysis of the anterior leg muscles

regaining power must be exercised, gently and patiently at first, but later very strenuously, to encourage the greatest possible restoration of power It should be remembered that power may improve partly as a result of recovery of the damaged nerve cells, but partly also from hypertrophy of muscle fibres that have escaped paralysis In this stage walking should be resumed whenever possible, if necessary with the aid of appliances, crutches, or sticks

Stage of residual paralysis. The disability in this stage can often be reduced either by the provision of suitable external appliances, or by operation

Appliances. The purpose of external appliances is to support joints that have lost their normal muscle control. They are required more often for the lower limbs and spine than for the upper limbs. The following are commonly prescribed: 1) spinal brace to support a weakened spine; 2) abdominal support, to check abdominal protrusion when the abdominal muscles are weak; 3) knee caliper (Fig. 78), to hold the knee extended in cases of quadriceps paralysis; 4) double below-knee steels (Fig. 79), to stabilise a flail ankle or foot; 5) single below-knee steel (lateral or medial) with ankle strap, to control varus or valgus deformity of the foot, 6) drop-foot spring (Fig. 80), to hold the foot up when the dorsiflexor muscles are paralysed

Operative treatment Two main groups of operations are available: 1) arthrodesis of joints; and 2) muscle transfers. *Arthrodesis* is a valuable method of stabilising joints that have lost their controlling muscles. It is particularly applicable to shoulder, elbow, wrist, spine, ankle, and foot. In *muscle transfer* operations the object is to use a healthy muscle to replace the function of one that is paralysed The method finds its chief application in the upper limb. Examples are the transfer of part of the pectoralis major muscle to replace the function of paralysed elbow flexors, transfer of wrist flexors to serve as extensors of the fingers, and transfer of a flexor digitorum sublimis tendon to replace a paralysed opponens muscle.

SPASTIC PARALYSIS

(Cerebral palsy)

Spastic paralysis usually arises at birth and becomes manifest within the first year of life. It is characterised by weakness, spasticity, and imperfect control of one or more of the limbs, and often also by mental deficiency. The primary lesion is in the cerebrum, and the neurological disturbance is of the upper motor neurone type

Cause. It is caused by destruction of the ganglion cells of the motor area of the cerebral cortex. Injury to the brain during difficult labour is usually responsible; less often the damage occurs after birth in consequence of haemorrhage or meningitis.

Pathology. Part of the motor cortex is replaced by areas of gliosis. There is degeneration of the pyramidal tracts.

Clinical features. There is often a history of difficult delivery. Later it is noticed that the child has difficulty in controlling the movements of the affected limbs, and there is delay in sitting up, standing, and walking. Commonly the upper and lower limbs of one side are affected (hemiplegia). Less often there is involvement of a single limb (monoplegia), of both lower limbs (paraplegia), or of all four limbs (diplegia ; quadriplegia). The trunk and face muscles may also be affected. *On examination* the features that are found constantly are weakness, spasticity, and imperfect voluntary control over movement. Additional features that are sometimes present are deformity, involuntary movements, and mental deficiency. These various features are best considered separately.

Weakness. There is no true paralysis such as the name of the disease might imply, but there may be fairly marked weakness of muscles. The weakness seldom affects all the muscles of a limb equally ; often there is marked muscle imbalance.

Spasticity. The muscles are " stiff " : they resist passive movement of the joints, but when steady pressure is applied for some time they slowly relax, allowing the joint to be moved. When the pressure is released the spasm immediately returns. The tendon reflexes are exaggerated.

Lack of voluntary control. This is a striking feature, especially in severe cases When the patient attempts to move a single group of muscles other groups contract at the same time.

Deformity. When spasm and muscle imbalance are pronounced they lead eventually to the development of fixed deformity. The stronger muscles hold the limb constantly in an unnatural position, and secondary adaptive changes take place in the muscles and peri-articular tissues. The commonest deformities in the upper limb are flexion contracture of the elbow, pronation deformity of the forearm, flexion of the wrist, and adduction of the thumb. In the lower limb the common deformities are adduction of the hip, flexion of the knee, and equinus of the ankle.

Involuntary movement. An occasional and troublesome feature is the occurrence of involuntary movements of athetoid type, which are increased by emotion.

Mental deficiency. Some impairment of mental capacity is often present, but by no means always. Lack of control of the facial and speech muscles makes the mental impairment seem worse than it actually is.

The severity of the disability varies widely from case to case. In the mildest examples the child is able to lead a normal active life with very little handicap, whereas in a severe case the patient is almost helpless.

Prognosis. Since an essential part of the brain is destroyed and cannot be replaced complete cure is impossible. All that can be hoped for is improvement. To achieve even this requires endless patience on the part of the patient and the attendants. In many cases little or no real improvement is gained even after prolonged treatment.

Treatment. Whenever possible treatment should be carried out at a special centre having adequate facilities and trained staff. The methods available are muscle education, corrective splinting, and operations on nerves, tendons, muscles, or bones.

Muscle education. The principle of muscle education is to teach the child to use the weaker muscles and so to increase their power and coordination. Repetitive rhythmic movements are a valuable form of exercise.

Corrective splinting. Splints or plasters are especially useful in overcoming the deformities induced by spastic muscles. Deformity is first corrected by gradual stretching of the contracted muscles, if necessary under anaesthesia. The limb is held by plaster in the over-corrected position for three months. Thereafter removable braces or splints may be used indefinitely to prevent recurrence of the deformity

Operative treatment Operations for spastic paralysis should be approached with caution, for the results are by no means uniformly satisfactory. Operation may be upon nerve, tendon, muscle, or bone. *Neurectomy :* The principle is to divide part or the whole of a nerve that supplies an over-acting spastic muscle. An example is division of the obturator nerve to overcome spastic adduction at the hip. *Tendon elongation :* Lengthening of the tendon of a spastic muscle reduces its mechanical advantage and thereby improves muscle balance An example is lengthening of the tendo calcaneus in a case of spastic equinus deformity.

Muscle transfer: In certain cases muscles can usefully be transferred to supplement the action of a weaker muscle. An example is transfer of the hamstring muscles to supplement the action of a weak quadriceps. *Arthrodesis of joints:* When skeletal growth is complete it is sometimes of benefit to fuse a joint in the position of function to overcome persistent deformity.

PERIPHERAL NERVE LESIONS

Disorders of the peripheral nerves come largely within the sphere of the neurologist, but the orthopaedic surgeon is concerned with lesions that have a mechanical basis and with those that lend themselves to reconstructive surgery

Pathology. Nerve trunks may be damaged by laceration, contusion, traction, compression, or ischaemia. According to its degree of severity, a nerve lesion may be classified as neurapraxia, axonotmesis, or neurotmesis. In *neurapraxia* the damage is slight and it causes only a transient physiological block. Recovery occurs spontaneously within a few weeks. In *axonotmesis* the internal architecture of the nerve is preserved, but the axons are so badly damaged that peripheral degeneration occurs. Recovery can occur spontaneously, but it depends upon regeneration of the axons and may take many months (about an inch a month is the usual speed of regeneration). In *neurotmesis* the structure of the nerve is destroyed by actual division or by severe scarring. Recovery is possible only after excision of the damaged section and end-to-end suture of the stumps.

In traction lesions and compression lesions the motor fibres tend to be affected more than the sensory, but in ischaemic lesions the sensory fibres are more vulnerable than the motor.

Clinical features. The effects of complete loss of conductivity of a nerve are motor, sensory, and autonomic. They are localised to the distribution of the nerve affected *Motor changes:* The muscles are paralysed and wasted. Changes also occur in the electrical reactions (reaction of degeneration) but they take between two and three weeks to develop *Sensory changes:* There is loss of cutaneous, deep, and postural sensibility. *Autonomic changes:* These include loss of sweating, loss of pilomotor response ("goose-skin") to cold, and temporary

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vasodilation with increased warmth, which, however, is followed later by vasoconstriction and coldness.

Diagnosis. Peripheral nerve lesions of the type under discussion must be distinguished from disorders of the central nervous system such as poliomyelitis, disseminated sclerosis, and syringomyelia; from peripheral neuritis; from the myopathies; and from hysteria.

Complications. Injury to a peripheral nerve containing sensory fibres is occasionally followed by severe burning pain in the distribution of the nerve. This is termed causalgia. It may be a troublesome complication, the only effective treatment of which is by sympathetic denervation of the limb.

Treatment. If a nerve is believed to be divided—for example, by a penetrating wound—it should be explored. If the diagnosis is confirmed the nerve should be sutured after resection of the scarred ends. The optimum time for definitive repair is three or four weeks after the injury.

In closed injuries complicated by nerve paralysis it is usually assumed that the nerve is in continuity and expectant treatment is adopted at first. If signs of beginning recovery are not observed within the expected time (calculated from the site of injury and length to be regenerated) exploration is advised.

When a nerve lesion has been caused by stretching, compression, or ischaemia, the essential principle of treatment is to ensure that the harmful conditions are relieved, if necessary by operation to free the nerve or remove a compressing agent.

The disability from permanent nerve paralysis can often be mitigated by muscle transfers or arthrodesis.

CHAPTER THREE

Neck and Cervical Spine

THE commonest orthopaedic cause of neck disorders is degeneration of a cervical intervertebral disc. This may lead to protrusion of part of the disc contents (prolapsed cervical disc), or it may give rise to secondary osteoarthritic changes in the intervertebral joints (cervical spondylosis). These conditions together make up a large proportion of the disabilities of the neck encountered in an orthopaedic out-patient department.

Disease of the cervical spine often interferes with the roots of the brachial plexus, causing radiating pain, muscle weakness, or sensory impairment in the upper limb. Indeed, the clinical importance of a cervical disorder often lies in its neurological effects rather than in the local lesion itself.

SPECIAL POINTS IN THE INVESTIGATION OF NECK COMPLAINTS

History

It is important to ascertain the relationship of the present symptoms to any previous neck disorder. Has there been any previous injury to the neck? Or a sudden jerk of the head that might have jarred the cervical spine? Is there a history of "stiff neck"—a common feature in the early stages of prolapsed cervical disc?

If pain in the upper limb is a prominent symptom, it is important to determine its exact distribution and its nature. Pain caused by pressure upon a nerve in the cervical region follows a clearly defined course which depends upon the particular nerve involved. It commonly [illegible] n into the forearm and hand, radiating [illegible] is often accompanied by paraesthesiae, [illegible] or "numbness." Pain referred down the limb from a lesion of the shoulder or humerus is more diffuse, and it seldom extends below the elbow.

Exposure

The patient must be stripped to the waist. Preferably he should stand, or he may sit upon a stool.

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Diagnosis. Peripheral nerve lesions of the type under discussion must be distinguished from disorders of the central nervous system such as poliomyelitis, disseminated sclerosis, and syringomyelia; from peripheral neuritis; from the myopathies; and from hysteria.

Complications. Injury to a peripheral nerve containing sensory fibres is occasionally followed by severe burning pain in the distribution of the nerve. This is termed causalgia. It may be a troublesome complication, the only effective treatment of which is by sympathetic denervation of the limb.

Treatment. If a nerve is believed to be divided—for example, by a penetrating wound—it should be explored. If the diagnosis is confirmed the nerve should be sutured after resection of the scarred ends. The optimum time for definitive repair is three or four weeks after the injury

In closed injuries complicated by nerve paralysis it is usually assumed that the nerve is in continuity and expectant treatment is adopted at first. If signs of beginning recovery are not observed within the expected time (calculated from the site of injury and length to be regenerated) exploration is advised.

When a nerve lesion has been caused by stretching, compression, or ischaemia, the essential principle of treatment is to ensure that the harmful conditions are relieved, if necessary by operation to free the nerve or remove a compressing agent

The disability from permanent nerve paralysis can often be mitigated by muscle transfers or arthrodesis.

throughout the cervical spine. Rotation occurs largely at the atlanto-axial joint, with a small range of movement at the other joints. It is important to find out whether movement causes pain and, if so, whether the pain is felt locally in the neck or whether it is referred down the upper limbs. It should be noted also whether movement is accompanied by audible or palpable crepitation.

Neurological Examination of Upper Limbs

This is an essential step in the investigation of the neck because cervical lesions so often interfere with the brachial plexus.

Muscular system. The muscles of the shoulder girdle, arm, forearm, and hand must be examined for wasting or fibrillation, a comparison being made on the two sides. The tone and power of each muscle group are then tested in turn and a comparison is made with the opposite limb.

Sensory system. Examine the patient's sensibility to touch and pin-prick. In appropriate cases test also the sensibility to deep stimuli, joint position, vibration, and heat and cold.

Reflexes. Compare on the two sides the biceps jerk (mainly C.6), the triceps jerk (mainly C.7), and the brachioradialis jerk (mainly C.6).

From the findings elicited it should be possible to determine whether there is a neurological disturbance and, if so, whether it is of upper or lower motor neurone type, and the identity of the roots, trunks, or branches involved.

Vascular Examination of the Upper Limb

The subclavian artery is sometimes interfered with by lesions of the neck. The efficiency of the circulatory system in each upper limb must therefore be determined. Judge and compare on the two sides the colour and warmth of the forearm, hand, and fingers Test and compare the radial pulses, first with the limb at rest, then with the shoulder depressed and the head rotated towards the side examined.

Extrinsic Causes of Neck Symptoms

Occasionally neck symptoms have their origin outside the neck itself. *Thus pain may be referred to the neck from the ears or throat. These sites should be examined routinely for evidence of disease.*

Neurological symptoms in the upper limb that might suggest the possibility of a neck disorder involving the brachial plexus may, in fact, have their origin in the shoulder or elbow, or at any point along the peripheral distribution of the nerve trunk

Radiographic Examination

Routine radiographs of the cervical spine include an antero-posterior and a lateral projection Additional projections are often required when it is desired to show a particular structure more clearly. For a study of the odontoid process of the axis a special antero-posterior projection is made through the open mouth Oblique projections are

Steps in Clinical Examination

A suggested routine for clinical examination of the neck is summarised in Table III.

TABLE III

ROUTINE CLINICAL EXAMINATION IN SUSPECTED DISORDERS OF THE NECK

1 Local examination of neck, with neurological and vascular survey of upper limbs

Inspection	Movements
Bone contours	Flexion-extension
Soft-tissue contours	Lateral flexion
Colour and texture of skin	Rotation
Scars or sinuses	? Pain on movement
	? Crepitation on movement
Palpation	**Neurological state of upper limb**
Skin temperature	Muscular system
Bone contours	Sensory system
Soft-tissue contours	Reflexes
Local tenderness	

Vascular state of upper limb

Colour
Temperature
Pulses

2 Examination of potential extrinsic sources of neck symptoms

Symptoms suggestive of a neck disorder may arise from the ears or throat. Neurological symptoms in the upper limb suggesting a neck disorder with involvement of the brachial plexus may arise from shoulder, elbow, or nerve trunks in their peripheral course

3 General examination

General survey of other parts of the body. Neck symptoms may be only one manifestation of a more widespread disease

Movements

The movements to be examined are flexion, extension, lateral flexion to right and left, and rotation to right and left. Flexion-extension movements occur mainly at the occipito-atlantoid joint but to some extent throughout the cervical spine. Lateral flexion takes place

INFANTILE TORTICOLLIS

(" Congenital " torticollis ; muscular torticollis)

Infantile torticollis is the commonest form of wry-neck. The head is tilted and rotated by contracture of the sternomastoid muscle of one side. Strictly this is not a true congenital lesion because it arises after birth.

Cause. This is uncertain. Probably there is interference with the blood supply of the sternomastoid muscle, caused by injury during birth.

Pathology. *In the established condition the affected muscle is* replaced by contracted fibrous tissue. In some cases contracture is known to have been preceded, in early infancy, by a tumour-like thickening of the muscle (" sternomastoid tumour "), the histology being that of muscle infarction and replacement by fibrous tissue.

Clinical features. The child, usually between 6 months and 3 years old, is noticed to hold its head on one side. *On examination*, the contracted muscle is felt as a tight cord The ear on *the affected side is approximated* to the corresponding shoulder. In long-established cases there is retarded development of the face on the affected side, with consequent asymmetry (Fig. 81).

FIG. 81
Infantile torticollis Note the tense cord-like left sternomastoid and the facial asymmetry

Diagnosis. The condition has to *be distinguished from other forms* of wry-neck, including structural deformities of the cervical spine, ocular torticollis, muscle spasm from a local inflammatory lesion such as infected glands, and psychogenic (hysterical) torticollis. *The important diagnostic features* are the history, the cord-like contracted muscle, and the facial asymmetry.

Treatment. If the condition is seen at the stage of " sternomastoid tumour," repeated stretching of the muscle under the supervision

essential for a proper investigation of the intervertebral foramina, and an oblique view of the lower cervical region is valuable in revealing the size and shape of a cervical rib. In cases of special difficulty stereoscopic films or tomographs may be helpful. If an intraspinal lesion is suspected, myelography is required.

CLASSIFICATION OF DISORDERS OF THE NECK AND CERVICAL SPINE

DEFORMITIES

Infantile torticollis
Congenital short neck
Congenital high scapula

INFECTIONS OF BONE

Tuberculosis of the cervical spine
Pyogenic infection of the cervical spine

ARTHRITIS OF THE SPINAL JOINTS

Ankylosing spondylitis
Osteoarthritis of the cervical spine

MECHANICAL DERANGEMENTS

Prolapsed intervertebral disc
Cervical rib
Cervical spondylolisthesis

TUMOURS

Benign and malignant tumours in relation to the cervical spine and nerve roots

MISCELLANEOUS

Cervical fibrositis

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Treatment. If the condition is seen at the stage of "sternomastoid tumour," repeated stretching of the muscle under the supervision

of a physiotherapist is effective. In established cases division of the contracted muscle at its lower attachment is advised.

CONGENITAL SHORT NECK

(Klippel-Feil syndrome)

This is an uncommon congenital malformation of the cervical spine characterised by short neck and limitation of head movements. The cause is unknown.

Pathology. The degree of abnormality varies widely. The bony deformity usually consists in fusion of two or more of the cervical vertebrae.

Clinical features. The neck appears short or absent. Movements of the head are restricted. *Radiographs* show the underlying bony abnormality, several vertebrae being fused together.

Treatment. There is no effective treatment.

CONGENITAL HIGH SCAPULA

(Sprengel's shoulder)

Congenital high scapula is an uncommon congenital deformity characterised by an abnormally high position and relative fixity of the scapula. The cause is unknown.

Pathology. The anomaly represents a failure of the scapula—originally a cervical appendage—to descend during development to its normal thoracic position The scapular muscles are ill developed and may be represented only by tough fibrous bands.

Clinical features. The scapula on one or both sides is abnormally high. Its attachments seem almost rigid and it does not rotate freely during abduction of the arm. The range of shoulder elevation is consequently impaired, but the functional disability is slight.

Treatment. The condition is usually best left alone The results of surgery have been disappointing.

TUBERCULOSIS OF THE CERVICAL SPINE

(Tuberculous cervical spondylitis)

Tuberculosis is far less common in the cervical spine than in the thoracic and lumbar regions. The general features of tuberculosis of bones and joints were described in Chapter II

Pathology. The organisms reach the cervical spine through the blood stream from a pre-existing focus—latent or overt—elsewhere in the body. The infection begins in the front of a vertebral body, or in an intervertebral disc (Fig. 82). Destruction of bone and intervertebral disc leads to anterior collapse with consequent cervical kyphosis (Fig. 83). The degree of destruction varies

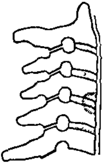

FIG 82

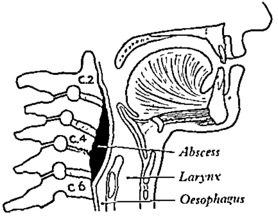

FIG 83

surfaces of the bodies of C.4 and C.5 have been eroded and the intervening disc is destroyed. Pus has collected behind the prevertebral fascia, forming a bulging retropharyngeal abscess.

widely, depending upon the virulence of the organism and the resistance of the patient. Formation of pus leads either to a retropharyngeal abscess (behind the prevertebral fascia), which may eventually point at the posterior margin of the sternomastoid, or, if the pus tracks posteriorly, to a sub-occipital abscess. The spinal cord may be damaged by direct pressure of an abscess, or by secondary thrombosis of the vessels of the cord.

Clinical features. The disease occurs mainly in children and young adults There is pain in the neck and occiput, aggravated by movement ; to relieve it the patient often supports his head in his hands In addition, one or more of the following symptoms may be present : difficulty in swallowing ; abscess or sinus at the side or back of the neck ; neurological symptoms from spinal cord dysfunction, the upper limbs being affected before the lower. *On examination* the head is held rigidly, often supported by the

hands. The cervical muscles stand out in spasm. One or more of the spinous processes may appear prominent, due to cervical kyphosis. There is local tenderness on firm palpation over the spinous processes. All movements of the head and neck are restricted, and cause pain if forced. An abscess may be present in the sub-occipital region, behind the sternomastoid, or behind the pharynx (see below). Associated tuberculous lesions elsewhere are common. *Radiographs* always show diminution of disc space, usually some destruction of bone (Fig. 83), and sometimes an abscess shadow. *Investigations :* The erythrocyte sedimentation rate is raised in the active stage.

Complications. *Retropharyngeal abscess :* This gives rise to difficulty in swallowing (dysphagia) Examination through the mouth shows the posterior wall of the pharynx bulged forwards in the midline Eventually the abscess may point behind the sternomastoid. If neglected, it may rupture into the pharynx.

Spinal cord dysfunction. If the spinal cord is affected there will be neurological signs (sensory, motor, and visceral) at and below the level of the lesion. Unless successfully treated, dysfunction may advance to complete paralysis

Diagnosis. Tuberculosis must be distinguished from other causes of pain and deformity of the neck, such as congenital malformations of the cervical vertebrae, infantile torticollis, cervical spondylolisthesis, skeletal deformities caused by previous injury, osteoarthritis, other infections of the spinal column, and ankylosing spondylitis. It must also be differentiated from other causes of retropharyngeal abscess and from other causes of spinal cord dysfunction. Important diagnostic features are the history (contact with tuberculosis, or tuberculous lesions elsewhere), the marked spasm of the neck muscles with restriction of all movements, abscess formation (the pus may yield tubercle bacilli), the radiographic findings, and the raised erythrocyte sedimentation rate.

Treatment. The principles of treatment are the same as for other forms of skeletal tuberculosis (p. 61). *General treatment* includes rest in a country hospital and chemotherapy in the form of streptomycin, para-amino-salicylic acid (PAS), and isonicotinic acid hydrazide (INAH), all three drugs being continued for six months if well tolerated. *Local treatment* is by rest for the

spine (in plaster bed, plaster case, or frame) until the disease is quiescent—often one to three years. Thereafter a collar-support is worn for several months. Operation may be required 1) to drain a retropharyngeal abscess that threatens to rupture or to cause asphyxia; 2) to decompress a spinal cord damaged by pressure of abscess or granuloma, if four weeks' conservative treatment by strict immobilisation fails to relieve the neurological disturbance; 3) in the quiescent stage, to fuse the affected region of the spine if it is judged to be unstable.

PYOGENIC INFECTION OF THE CERVICAL SPINE

(Pyogenic cervical spondylitis)

Infection of the cervical vertebrae or intervertebral discs with pyogenic organisms is uncommon.

Cause. It is caused by infection with the streptococcus, staphylococcus or pneumococcus, or occasionally with other bacteria.

Pathology. The organisms reach the spinal column by the general blood stream (from a septic focus elsewhere), by the spinal venous plexus (from a focus in the pelvis), or by lymphatic channels (from a local infection, for instance in the pharynx). As in tuberculous spondylitis, there is destruction of bone and intervertebral disc, with or without abscess formation. The spinal cord may be damaged by direct pressure or by thrombosis.

Clinical features. The onset is usually acute or subacute, with pyrexia. The clinical features are like those of tuberculous spondylitis (p. 126), but the course is more rapid. A suppurative process elsewhere in the body (for instance, in the pharynx or pelvis) is usually present. *Radiographs* show local osteoporosis or erosion of bone, diminution of disc space, and sometimes subligamentous new bone formation. *Investigations:* The erythrocyte sedimentation rate is raised and polymorphonuclear leucocytosis is to be expected.

Diagnosis. The condition must be distinguished from tuberculous spondylitis, which it may resemble very closely, and from the other conditions mentioned in the diagnosis of tuberculous spondylitis (p. 128) A history of pre-existing septic focus, the relatively rapid onset and course, with pyrexia and leucocytosis, and identification of the causal organism in pus, are the main diagnostic criteria.

Treatment. Penicillin or another appropriate antibiotic drug should be given systemically in large doses. The cervical spine must be immobilised in a plaster bed, plaster case, or frame; sometimes continuous head traction is required for relief of spasm. Abscesses should be drained, especially if the spinal cord is threatened. Later, a collar-support may be required, but spontaneous fusion of the affected vertebrae usually makes operative fusion unnecessary.

ANKYLOSING SPONDYLITIS

Ankylosing spondylitis creeps up the spine from below. Only in about a third of the cases does it reach the cervical region. The general features of the disease as a whole will be described in the chapter on the spine (p. 165). The condition is considered here only in so far as it affects the neck.

Pathology. There is chronic inflammation of the intervertebral joints, leading eventually to bony ankylosis if the disease progresses.

Clinical features. During the active phase of neck involvement there is a diffuse aching pain in the neck, with increasing stiffness. Eventually there may be total rigidity of the neck, but commonly a small range of occipito-atlantoid and of atlanto-axial movement is spared. The changes typical of ankylosing spondylitis will be found in the thoracic and lumbar regions. *Radiographic examination:* In the late stages there is obvious bony fusion of the cervical vertebrae. *Investigations:* The erythrocyte sedimentation rate is raised during the active phase.

Treatment. Deep x-ray therapy retards the progress of the disease and relieves pain. Any movement that remains should be encouraged by active exercises.

OSTEOARTHRITIS OF THE CERVICAL SPINE

(Cervical spondylosis)

Degenerative changes are common in the cervical spine. Beginning in the intervertebral discs, they affect the posterior intervertebral joints secondarily, causing pain and stiffness of the neck, sometimes with referred symptoms in an upper limb.

Cause. The primary degenerative changes are often initiated by injury. In other cases the condition is simply a manifestation of the widespread degenerative changes that occur with increasing age.

Pathology. The changes affect first the central intervertebral joints (between the vertebral bodies) and later the posterior intervertebral (facet) joints. In the central joints there is degeneration and consequent narrowing of the intervertebral disc, and bone reaction at the joint margins leads to the formation of

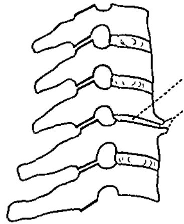

FIG. 84

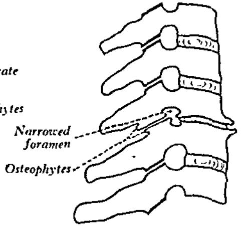

FIG. 85

Osteoarthritis of the cervical spine At first there is simply degeneration and narrowing of the intervertebral disc, with the formation of osteophytes anteriorly (Fig 84). Later, the posterior or facet joints are affected: the articular cartilage is worn away and marginal osteophytes may encroach upon the intervertebral foramen (Fig 85)

osteophytes (Fig. 84) In the posterior intervertebral joints the changes are those of osteoarthritis in any diarthrodial joint—namely, wearing away of the articular cartilage and osteophyte formation (lipping) at the joint margins (Fig. 85).

Secondary effects. Osteophytes commonly encroach upon the intervertebral foramina, reducing the space for transmission of the cervical nerves (Fig. 85). If the restricted space in a foramen is still further reduced by traumatic oedema of the contained soft tissues, manifestations of nerve pressure are likely to occur. Exceptionally, the spinal cord itself may suffer damage from encroachment of osteophytes within the spinal canal.

Clinical features. The symptoms are in the neck, or in the upper limb, or both. *Neck* symptoms consist chiefly of aching pain in the back of the neck or in the trapezius area, and "grating" on movement. Usually slight, they are liable to periodic exacerbations, probably from unremembered strains. *Upper limb* symptoms are caused by interference with one or more of the cervical nerves in their foramina. They occur as a more noticeable incident of limited duration (weeks or months) punctuating the chronic mild discomfort in the neck. The main feature is radiating pain along the course of the affected nerve or nerves, often reaching the digits. There may also be paraesthesiae, in the form of "tingling" or "pins and needles," in the hand. Noticeable muscle weakness is exceptional. *On examination*, the neck may be slightly kyphotic. The posterior cervical muscles may be somewhat tender but they are not in spasm. Movements are not noticeably diminished except during acute exacerbations or when the degenerative changes are very advanced. Audible crepitation on movement is common. In the upper limb objective findings are usually slight or absent, for the nerve pressure is seldom great enough to produce well defined objective neurological signs (compare prolapsed intervertebral disc). Thus demonstrable motor weakness or sensory impairment is exceptional. Depression of one or more of the tendon reflexes is, however, fairly common *Radiographic examination:* There is narrowing of the intervertebral disc space, with formation of osteophytes at the vertebral margins, especially anteriorly (Fig 86).

FIG. 86
Osteoarthritis of cervical spine Note the narrowed intervertebral space, with marginal osteophyte formation, at C.5-6 and at C 6-7

Diagnosis. Distinction has to be made 1) from other causes of neck pain, and 2) from other causes of upper limb pain

Other causes of neck pain : These include prolapsed cervical disc, tuberculous or pyogenic infection, and fibrositis.

Other causes of upper limb pain : These are as follows : *Central lesions*—Tumours involving the spinal cord or its roots ; cervical spondylolisthesis. *Plexus lesions*—Tumours at the thoracic inlet (Pancost) ; cervical rib ; prolapsed intervertebral disc. *Shoulder lesions* with radiating pain in the upper arm. *Skeletal lesions* such as a tumour, infection, or Paget's disease of a bone of the upper extremity. *Elbow lesions,* such as "tennis elbow" or arthritis *Distal nerve lesions* such as friction "neuritis" of the ulnar nerve at the elbow or of the median nerve in the carpal tunnel.

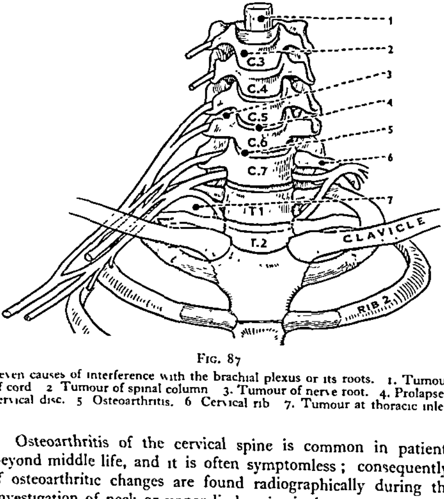

FIG. 87

Seven causes of interference with the brachial plexus or its roots. 1. Tumour of cord 2 Tumour of spinal column 3. Tumour of nerve root. 4. Prolapsed cervical disc. 5 Osteoarthritis. 6 Cervical rib 7. Tumour at thoracic inlet.

Osteoarthritis of the cervical spine is common in patients beyond middle life, and it is often symptomless ; consequently, if osteoarthritic changes are found radiographically during the investigation of neck or upper limb pain, it does not necessarily

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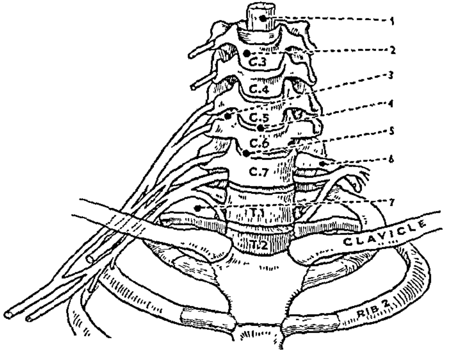

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follow that the symptoms are caused by the arthritis. The diagnosis is at best presumptive, never unequivocal; before it is justified, other causes of similar symptoms must be excluded by careful investigation (Fig. 87).

Treatment. There is a strong tendency for the symptoms to subside spontaneously, though they may persist for many months. It is doubtful whether treatment hastens recovery. Palliative measures include mild analgesic drugs, physiotherapy (radiant heat, short-wave diathermy, massage, or exercises), and, in the worst cases, a supportive collar of plaster or plastic.

In the exceptional cases in which the spinal cord is constricted, decompression by laminectomy may be required.

PROLAPSED CERVICAL DISC

Displacement of intervertebral disc material in the cervical spine is much less common than it is in the lumbar region. Even so, examples are encountered fairly frequently. The condition is characterised by pain and stiffness in the neck, often with neurological manifestations in the upper limb and sometimes with signs of spinal cord compression.

Cause. Injury is a predisposing factor, though a history of injury cannot be obtained in every case. Probably an intrinsic change in the substance of the disc makes it prone to rupture and displacement.

Pathology. The disc between C.5-6 and that between C.6-7 are those most frequently affected. Part of the gelatinous nucleus *pulposus protrudes through a rent in the annulus fibrosus at its* weakest part, which is postero-lateral. If slight, the protrusion bulges the pain-sensitive posterior longitudinal ligament, causing local pain in the neck. If large, the protrusion herniates through the posterior ligament and may impinge upon the nerve leaving the spinal canal at that level (lateral prolapse) (Fig. 88) or occasionally upon the spinal cord itself (central prolapse) (Fig. 89). Healing is probably by shrinkage and fibrosis of the extruded material rather than by its reposition within the disc. *Secondary effects:* Prolapse of a disc accelerates its degeneration and predisposes to the development of osteoarthritis in later years.

Clinical features. *Central protrusions:* These lead to manifestations of spinal cord compression and may be confused with spinal cord tumours or other central neurological disorders. They fall within the province of the neurosurgeon rather than the orthopaedic surgeon. *Lateral protrusions:* The characteristic clinical picture is as follows. The patient sustains an injury to

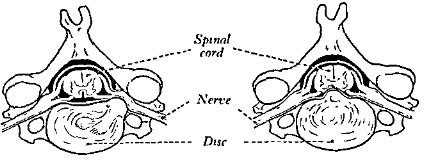

FIG 88 FIG. 89

Prolapsed cervical disc Figure 88 shows a lateral prolapse, with compression of the issuing nerve Figure 89 shows the much less common central prolapse, with impingement upon the spinal cord

the neck—often a jarring or twisting strain—which may seem slight at the time and may cause no immediate effects. Hours or days later there is a rapid development of acute "stiff neck," with severe pain made worse by coughing or similar strains. Hours or days later still, the pain begins to radiate over the shoulder and throughout the length of the upper limb; it is felt strictly in the course of a cervical nerve. Paraesthesiae are felt in the digits. *On examination,* there is limitation of certain neck movements by pain, but movement in at least one direction (often lateral flexion away from the affected side) is free. In the upper limb there is a full range of joint movements. There are slight muscle wasting and slight sensory impairment in the distribution of a cervical nerve The corresponding tendon reflex (biceps jerk in C.5-6 lesions; triceps jerk in C.6-7 lesions) is depressed or absent.

Variations. The characteristic features described are not always present. Variations are common. Thus a history of injury is not always obtainable. The symptoms may be confined to the neck, the upper limb being spared; or they may be confined entirely to the upper limb. Motor changes (wasting and weakness) may

follow that the symptoms are caused by the arthritis. The diagnosis is at best presumptive, never unequivocal; before it is justified, other causes of similar symptoms must be excluded by careful investigation (Fig. 87).

Treatment. There is a strong tendency for the symptoms to subside spontaneously, though they may persist for many months It is doubtful whether treatment hastens recovery. Palliative measures include mild analgesic drugs, physiotherapy (radiant heat, short-wave diathermy, massage, or exercises), and, in the worst cases, a supportive collar of plaster or plastic.

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features. *Central protrusions:* These lead to manifestations of spinal cord compression and may be confused with spinal cord tumours or other central neurological disorders. They fall within the province of the neurosurgeon rather than the orthopaedic surgeon. *Lateral protrusions:* The characteristic clinical picture is as follows. The patient sustains an injury to

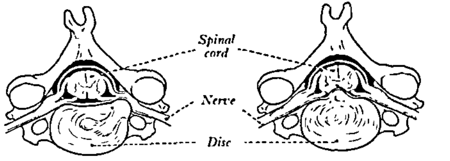

FIG 88 FIG. 89

Prolapsed cervical disc. Figure 88 shows a lateral prolapse, with compression of the issuing nerve. Figure 89 shows the much less common central prolapse, with impingement upon the spinal cord.

the neck—often a jarring or twisting strain—which may seem slight at the time and may cause no immediate effects. Hours or days later there is a rapid development of acute " stiff neck," with severe pain made worse by coughing or similar strains. Hours or days later still, the pain begins to radiate over the shoulder and throughout the length of the upper limb; it is felt strictly in the course of a cervical nerve. Paraesthesiae are felt in the digits. *On examination*, there is limitation of certain neck movements by pain, but movement in at least one direction (often lateral flexion away from the affected side) is free. In the upper limb there is a full range of joint movements. There are slight muscle wasting and slight sensory impairment in the distribution of a cervical nerve. The corresponding tendon reflex (biceps jerk in C 5-6 lesions; triceps jerk in C.6-7 lesions) is depressed or absent.

Variations. The characteristic features described are not always present Variations are common. Thus a history of injury is not always obtainable. The symptoms may be confined to the neck, the upper limb being spared; or they may be confined entirely to the upper limb Motor changes (wasting and weakness) may

follow that the symptoms are caused by the arthritis. The diagnosis is at best presumptive, never unequivocal; before it is justified, other causes of similar symptoms must be excluded by careful investigation (Fig. 87).

Treatment. There is a strong tendency for the symptoms to subside spontaneously, though they may persist for many months. It is doubtful whether treatment hastens recovery. Palliative measures include mild analgesic drugs, physiotherapy (radiant heat, short-wave diathermy, massage, or exercises), and, in the worst cases, a supportive collar of plaster or plastic.

In the exceptional cases in which the spinal cord is constricted, decompression by laminectomy may be required.

PROLAPSED CERVICAL DISC

Displacement of intervertebral disc material in the cervical spine is much less common than it is in the lumbar region. Even so, examples are encountered fairly frequently. The condition is characterised by pain and stiffness in the neck, often with neurological manifestations in the upper limb and sometimes with signs of spinal cord compression.

Cause. Injury is a predisposing factor, though a history of injury cannot be obtained in every case. Probably an intrinsic change in the substance of the disc makes it prone to rupture and displacement.

Pathology. The disc between C.5-6 and that between C.6-7 are those most frequently affected. Part of the gelatinous nucleus pulposus protrudes through a rent in the annulus fibrosus at its weakest part, which is postero-lateral. If slight, the protrusion bulges the pain-sensitive posterior longitudinal ligament, causing local pain in the neck. If large, the protrusion herniates through the posterior ligament and may impinge upon the nerve leaving the spinal canal at that level (lateral prolapse) (Fig. 88) or occasionally upon the spinal cord itself (central prolapse) (Fig. 89). Healing is probably by shrinkage and fibrosis of the extruded material rather than by its reposition within the disc. *Secondary effects:* Prolapse of a disc accelerates its degeneration and predisposes to the development of osteoarthritis in later years.

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FIG. 90
Plastic collar for immobilising the cervical spine in severe cases of prolapsed disc or osteoarthritis.

CERVICAL RIB

A cervical rib is a congenital over-development, bony or rous, of the costal process of the seventh cervical vertebra. It y exist without causing symptoms, or it may cause neurological vascular disturbances in the upper limb.

The cause of the congenital error is unknown. The ten- to dropping of the shoulder girdle that occurs in adult life held responsible for the onset of symptoms.

The over-developed costal process may be unilateral bilateral. It may be of any size from a small bony protrusion, with a fibrous extension, to a complete supernumerary rib. he subclavian artery and the lowest trunk of the brachial plexus h over the rib. In a proportion of cases the nerve trunk suffers e at the site of pressure against the rib ; this accounts for e neurological manifestations. The less common vascular nanges are probably similarly accounted for by local damage to e subclavian artery, though this explanation has been disputed.

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be marked, sometimes amounting to almost complete paralysis of a muscle or a group of muscles; or, on the other hand, they may be absent. Similarly, wide variations in the degree of sensory impairment are noted.

Radiographs characteristically show normal appearances in the first attack, but narrowing of one of the disc spaces (usually C.5-6 or C.6-7) is often demonstrable. Such narrowing denotes previous disc degeneration; it cannot be explained simply by extrusion of disc substance because the extruded matter forms only a small proportion of the total volume of the disc.

Diagnosis.[1] Prolapsed cervical disc has to be differentiated 1) from other causes of neck pain, and 2) from other causes of upper limb pain (Fig. 87). The main conditions that may be confused with it are the same as those listed in the differential diagnosis of cervical osteoarthritis (p. 132). Diagnosis is presumptive and never unequivocal (unless confirmed at operation). A confident diagnosis is justified only when a suggestive history is associated with the signs of a lesion of a single cervical nerve, and provided always that other possible causes have been excluded by careful investigation. Diagnostic myelography is not justified unless a spinal tumour is seriously suspected.

Relationship between prolapsed cervical disc and cervical osteoarthritis. The clinical features of the two conditions are similar. Distinction is difficult if the radiographs show arthritic changes, for the arthritis may be only incidental and itself symptomless. Nerve pressure is probably greater in prolapsed disc than in osteoarthritis; consequently the symptoms tend to be more clearly defined and the objective signs more marked. Fortunately differentiation is not important in practice, for the treatment of both conditions is similar.

Course.[1] There is a strong tendency to spontaneous recovery, but symptoms often persist with decreasing severity for as long as six months or more

Treatment.[1] This depends upon the nature and severity of the individual case. When the symptoms are slight no treatment other than perhaps a mild analgesic drug is required. In the more severe cases treatment is advisable, especially in the early acute

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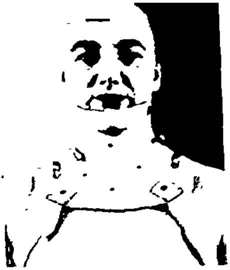

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Clinical features. Cervical rib is often symptomless. When symptoms occur, they usually begin during early adult life. They may be neurological, vascular, or combined.

Neurological manifestations. The sensory symptoms are pain and paraesthesiae in the forearm and hand, most marked towards the medial (ulnar) side. The motor symptoms include increasing weakness of the hand, with difficulty in carrying out the finer movements. *On examination*, there is usually an area of sensory impairment—sometimes complete anaesthesia—in the forearm or

FIG. 91
Cervical rib Typical appearance of a small supernumerary rib This one caused severe neurological symptoms and signs A cervical rib is shown best by an oblique radiograph such as this

hand The affected area does not correspond in distribution to any of the peripheral nerve trunks. There is wasting of the muscles of the thenar eminence (characteristically the flexor pollicis brevis is spared) or of the interosseous and hypothenar muscles.

Vascular manifestations The changes that have been observed range from dusky cyanosis of the forearm and hand to gangrene of the fingers The radial pulse may be weak or absent

Radiographs show the abnormal rib · if small, it is seen best in oblique projections (Fig. 91).

Diagnosis. Radiographic demonstration of a cervical rib does not prove that it is the cause of symptoms. The condition has to be distinguished 1) from other causes of pain in the forearm and hand, 2) from other causes of muscle wasting in the hand, and 3) from other causes of peripheral vascular changes in the upper limb.

Other causes of pain in the forearm and hand (Fig. 87). The important alternative causes are as follows: *Central lesions*—Tumours involving the spinal cord or its roots; cervical spondylolisthesis. *Plexus lesions*—Tumours at the thoracic inlet (Pancost); prolapsed cervical disc; osteoarthritis of the cervical spine. *Distal nerve lesions*—Friction "neuritis" of the ulnar nerve at the elbow; pressure upon the median nerve in the carpal tunnel.

Other causes of muscle wasting in the hand. These include the following: *Central lesions*—Syringomyelia; tumour of spinal cord; poliomyelitis; progressive muscular atrophy; cervical spondylolisthesis. *Plexus lesions*—Tumours at the thoracic inlet (Pancost); prolapsed cervical disc (especially at C.7-T.1). *Distal nerve lesions*—Friction "neuritis" of the ulnar nerve at the elbow; pressure upon the median nerve in the carpal tunnel; toxic neuritis. *Muscle lesions*—Muscular dystrophy.

Other causes of upper limb peripheral vascular lesions. These include embolism and Raynaud's disease.

The diagnosis of symptomatic cervical rib depends upon the detection of the characteristic neurological signs or vascular disturbance in association with a demonstrable supernumerary rib. Prolapsed intervertebral disc at C.7-T.1 gives a similar clinical picture, but in that condition there is a strong tendency to spontaneous recovery.

Treatment. This depends upon the severity of the subjective and objective manifestations. In mild cases physiotherapy, to improve the tone of the elevator muscles of the shoulder girdle, is adequate. But if the neurological or vascular signs are well marked, and especially if they are increasing, operation is advisable. First the scalenus anterior muscle is divided. If this does not demonstrably release the lowest nerve trunk from constricting pressure the scalenus medius should be divided and the abnormal rib removed.

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slow redisplacement months or years after the initial injury (Fig. 94).

In all types the upper segment is displaced forwards in relation to the lower. The spinal canal becomes progressively more flexed and narrowed, and there is always a grave risk of compression of the spinal cord.

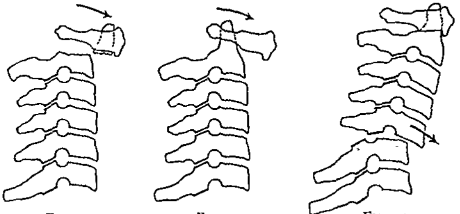


FIG. 92 FIG 93 FIG. 94

Three types of cervical spondylolisthesis or spontaneous subluxation. Figure 92—Displacement of atlas with odontoid peg permitted by congenital or post-traumatic non-fusion of the odontoid with the axis. Figure 93—Displacement of atlas on axis, from softening of the transverse ligament of the atlas. Figure 94—Subluxation of the fifth on the sixth cervical vertebra due to instability of the intervertebral joint after previous injury.

Clinical features. In the post-inflammatory type the patient is often a child with an acute infective lesion in the ear, throat, or neck. There is complaint of "stiff neck." The head is held rigidly, the cervical muscles being in spasm. In subluxation from congenital or post-traumatic instability there are discomfort and stiffness in the neck, and flexion deformity is apparent on examination.

Complications. In all types the complication to be feared is compression of the spinal cord. This is indicated clinically by the development of neurological manifestations. The first symptoms appear in the upper limbs and consist of root pain, paraesthesiae, motor weakness, or sensory impairment. Eventually, increasing cord compression leads to spastic paralysis below the level of the lesion, and to bladder and bowel dysfunction.

FIRST RIB SYNDROME (Scalenus syndrome)

Occasionally the neurological manifestations characteristic of cervical rib occur in the absence of a demonstrable skeletal abnormality They have been ascribed by some surgeons to trapping of nerves between the first rib and the clavicle (costo-clavicular compression), and by others to stretching of the lowest trunk of the brachial plexus over the normal first rib. Though this may be the correct explanation in a few cases, it is probable that most are examples of prolapsed intervertebral disc between C.7 and T.1. Treatment should therefore be conservative at first, as for prolapsed disc. Gradual improvement would support that diagnosis But if the symptoms persist with undiminished intensity for a long time a true " first rib syndrome " is probably the cause. In that event the correct treatment is to excise an appropriate segment of the first rib.

CERVICAL SPONDYLOLISTHESIS

(Spontaneous subluxation of the cervical spine)

In cervical spondylolisthesis there is spontaneous displacement, usually forwards, of one cervical vertebra upon the next below it. **Causes and pathology.** There are three types, caused by 1) congenital failure of fusion of the odontoid process with the axis, 2) inflammatory softening of the transverse ligament of the atlas, and 3) *instability from previous injury*

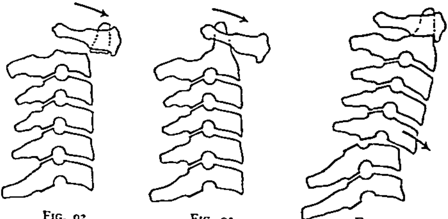
Congenital non-fusion of odontoid process. Occasionally the odontoid peg fails to fuse with the axis by bone, being attached only by fibrous tissue Under the constant stress of superimposed weight the fibrous bond slowly stretches, allowing the odontoid peg, and with it the atlas and skull, to slide gradually forwards upon the axis (Fig. 92). A similar condition may be caused by fracture of the odontoid.

Inflammatory softening of the transverse ligament of the atlas. In this type the underlying cause is an inflammatory lesion in the upper part of the neck, such as an infection of the ear, throat, or glands. There is osteoporosis of the atlas, with softening of the transverse ligament In consequence the atlas is able to slide forwards upon the axis (Fig 93).

Post-traumatic instability. A traumatic fracture-dislocation or subluxation may cause permanent instability, with a liability to

slow redisplacement months or years after the initial injury (Fig. 94).

In all types the upper segment is displaced forwards in relation to the lower. The spinal canal becomes progressively more flexed and narrowed, and there is always a grave risk of compression of the spinal cord.



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Treatment. This depends upon the underlying cause and upon whether or not neurological disturbance is present. *Inflammatory type* (usually children): The displacement is reduced by head traction, which is continued for two weeks. Thereafter a hyper-extension plaster jacket is worn for two months. *Congenital or post-traumatic instability*: If subluxation is not complicated by neurological disturbance, treatment may be expectant (observation only), by supportive plastic collar (Fig. 90), or by local fusion of the spine, according to the severity of the displacement and of the local symptoms. If neurological disturbance is present, treatment is by preliminary skull traction to reduce the displacement, followed by operative fusion of the affected segments of the spine.

TUMOURS IN RELATION TO THE CERVICAL SPINE AND NERVE ROOTS

Classification and pathology. These tumours are best classified according to their site and nature. *Tumours of the spinal cord or meninges*: These include meningioma, intradural neurofibroma, and, more rarely, glioma. *Tumours of nerves*: The important one is neurofibroma, which may be single or multiple. If it arises from a nerve in the intervertebral foramen it may grow inwards to compress the spinal cord and outwards towards the surface (" dumb-bell " tumour) *Tumours of the bony spinal column*: Benign tumours of the spinal column are rare Malignant tumours may be primary (sarcoma, multiple myeloma) or metastatic (carcinoma, sarcoma) *Tumours at the thoracic inlet*: Pancost's tumour at the thoracic inlet (usually an apical lung carcinoma) invades the base of the neck and is appropriately included here. Metastatic tumours in this area are usually lymph gland metastases

Clinical features. The effects of these tumours vary according to their site. Broadly, the effects may be placed in three groups: 1) spinal cord compression, 2) local destruction and collapse of cervical vertebrae, and 3) interference with the brachial plexus.

Spinal cord compression Interference with the function of the spinal cord may be caused by tumours of the cord itself or of its meninges, by tumours of nerves (neurofibroma), or by tumours of the bony spinal column The clinical manifestations depend upon the exact location of the tumour Typically, root pains at

the level of the lesion are followed by lower motor neurone changes at the same level and by progressive upper motor neurone paralysis and visceral dysfunction below the lesion.

Destruction and collapse of cervical vertebrae. The commonest cause is a metastatic carcinoma. The clinical features are local pain and, usually, flexion deformity. The spinal cord or the issuing cervical nerves may be involved, with corresponding signs of peripheral nerve disturbance or spinal cord compression.

Interference with the brachial plexus. Nerves forming the plexus may be involved by tumours of the nerves themselves (neurofibroma), by bone tumours, or by tumours at the thoracic inlet. The predominant features are severe pain along the course of the nerve or nerves affected (neck or upper limb) and increasing motor and sensory impairment in the distribution of the nerves.

Radiographic features. Plain radiographs will usually help in discovering a tumour arising in the bones of the spinal column or eroding the bones from outside. They may also reveal a tumour at the thoracic inlet. Myelography is helpful in the diagnosis of intraspinal tumours. Radiographs of the chest may reveal an apical lung tumour or pulmonary metastases. Radiographs of other bones are required if a widespread skeletal tumour such as multiple myeloma is suspected.

Investigations. In appropriate cases the following may be informative: Urine tests for Bence-Jones proteose (for multiple myeloma or leukæmia), blood counts (leukæmia), acid phosphatase estimation (prostatic metastases), and marrow biopsy (leukæmia, lymphadenoma). Careful search should be made for a possible primary tumour elsewhere in the body.

Diagnosis. Tumours must be differentiated 1) from other localised lesions of the spinal cord (syringomyelia, hæmatomyelia, cervical spondylolisthesis); 2) from other causes of local pain in the neck with or without cervical vertebral collapse (tuberculous spondylitis, pyogenic spondylitis, osteoarthritis, cervical spondylolisthesis); and 3) from other causes of radiating pain and muscle weakness in the upper limb (prolapsed cervical disc, osteoarthritis of the cervical spine, cervical rib) (Fig. 87). Important diagnostic features that should always suggest the possibility of a tumour are a slow insidious onset and steady relentless progress without remission.

Treatment. This depends upon the nature and site of the tumour. In general, the choice lies between excision, for those that are amenable to it, and palliative deep x-ray therapy for malignant tumours that cannot be removed. Hormone therapy (stilboestrol or testosterone) is appropriate for certain prostatic or breast metastases.

CERVICAL FIBROSITIS

(Muscular rheumatism)

The existence of fibrositis has been questioned, and it is admitted that it cannot be supported on pathological grounds. Nevertheless, the term " cervical fibrositis " is a useful label for a fairly clear-cut clinical condition characterised by pain and tenderness in the muscles at the base of the neck, but without other objective signs.

Cause. This is unknown.

Pathology. There are no demonstrable pathological changes So-called fibrositic nodules cannot be identified histologically.

Clinical features. There is pain at the base of the neck posteriorly, with radiation towards the back of the shoulder on one or both sides The pain is inconstant and varies in severity, often with climatic changes. There may be complaint of similar pain in other muscle groups *On examination*, the only sign is local tenderness on firm pressure over the affected muscles or on squeezing them between the fingers The trapezius and levator scapulae are most commonly affected. Radiography and other investigations show normal findings.

Diagnosis. Other causes of neck pain must be excluded by a careful history, clinical examination, and radiography.

Treatment. Often reassurance alone is required. When discomfort is marked, physiotherapy in the form of local heat and deep massage hastens spontaneous relief.

CHAPTER FOUR

Trunk and Spine

PAIN in the back is the commonest symptom encountered in orthopaedic practice. Indeed, if accident cases are excluded, it accounts for nearly a third of all orthopaedic out-patient attendances.

When this huge mass of material is sifted and categorised it is found that the cases fall into two broad groups. In the first group clear-cut physical signs, with or without distinctive radiographic changes or other abnormal findings, allow a precise determination of the nature of the lesion and of its site. The diagnosis is positive, and rational treatment can be applied. In the second group, almost as large as the first, there are no abnormal findings on clinical and radiographic examination. Diagnosis is largely a matter of conjecture, and treatment is empirical. For want of more accurate knowledge these rather vague and unsatisfactory cases are generally classed as "chronic ligamentous strain" or "postural back pain."

Lumbar back pain is often accompanied by radiating pain in the buttock, thigh, or leg, usually on one side but occasionally on both. This pain is generally referred to as sciatica, though the term should strictly be reserved for pain in the distribution of the sciatic nerve. It should be noted that sciatica is often a much more disturbing symptom of back disorders than the back pain itself, which indeed may be slight or transient.

SPECIAL POINTS IN THE INVESTIGATION OF BACK AND SCIATIC SYMPTOMS

History

Special attention should be paid to the mode of onset of the symptoms, whether they are periodic or constant, whether they are getting worse or better, and what relieves them or aggravates them. The precise location of the back pain and its character should be determined. The patient should be asked to what he attributes his

Treatment. This depends upon the nature and site of the tumour. In general, the choice lies between excision, for those that are amenable to it, and palliative deep x-ray therapy for malignant tumours that cannot be removed. Hormone therapy (stilboestrol or testosterone) is appropriate for certain prostatic or breast metastases.

CERVICAL FIBROSITIS

(Muscular rheumatism)

The existence of fibrositis has been questioned, and it is admitted that it cannot be supported on pathological grounds. Nevertheless, the term " cervical fibrositis " is a useful label for *a fairly clear-cut clinical condition characterised by pain and* tenderness in the muscles at the base of the neck, but without other objective signs.

Cause. This is unknown.

Pathology. There are no demonstrable pathological changes. So-called fibrositic nodules cannot be identified histologically.

Clinical features. There is pain at the base of the neck posteriorly, with radiation towards the back of the shoulder on one or both sides. The pain is inconstant and varies in severity, often with climatic changes. There may be complaint of similar pain in other muscle groups. *On examination*, the only sign is local tenderness on firm pressure over the affected muscles or on squeezing them between the fingers. The trapezius and levator scapulae are most commonly affected. Radiography and other investigations show normal findings.

Diagnosis. Other causes of neck pain must be excluded by a careful history, clinical examination, and radiography.

Treatment. Often reassurance alone is required When discomfort is marked, physiotherapy in the form of local heat and deep massage hastens spontaneous relief.

CHAPTER FOUR

Trunk and Spine

PAIN in the back is the commonest symptom encountered in orthopaedic practice. Indeed, if accident cases are excluded, it accounts for nearly a third of all orthopaedic out-patient attendances.

When this huge mass of material is sifted and categorised it is found that the cases fall into two broad groups. In the first group clear-cut physical signs, with or without distinctive radiographic changes or other abnormal findings, allow a precise determination of the nature of the lesion and of its site. The diagnosis is positive, and rational treatment can be applied. In the second group, almost as large as the first, there are no abnormal findings on clinical and radiographic examination. Diagnosis is largely a matter of conjecture, and treatment is empirical. For want of more accurate knowledge these rather vague and unsatisfactory cases are generally classed as "chronic ligamentous strain" or "postural back pain."

Lumbar back pain is often accompanied by radiating pain in the buttock, thigh, or leg, usually on one side but occasionally on both. This pain is generally referred to as sciatica, though the term should strictly be reserved for pain in the distribution of the sciatic nerve. It should be noted that sciatica is often a much more disturbing symptom of back disorders than the back pain itself, which indeed may be slight or transient.

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takes the form of a vague diffuse ache, ill defined in its distribution, it is more likely to be a "referred" pain originating in a disordered joint or ligament.

Exposure

The patient should be stripped completely, except for short underpants or a special pelvic slip and, in women, a brassiere.

Steps in Routine Examination

A suggested plan for the routine clinical examination of the back is summarised in Table IV.

Movements of the Spinal Column and Related Joints

The spinal column. The joints of the spinal column must necessarily be considered as a group, for it is impracticable to study the movement of each one independently. The movements to be examined are flexion, extension, lateral flexion to right and left, and rotation to right and left. It should be noted particularly whether the spinal muscles go into protective spasm when movement is attempted. *Flexion :* Instruct the patient to stretch his fingers towards his toes, keeping the knees straight. It is important to judge what proportion of the movement occurs at the spine and how much is contributed by hip

FIG 95

FIG 96

Figure 95—Normal flexion of lumbar spine
Figure 96—Apparent or false flexion due entirely to movement at the hips, the hamstrings being unusually lax. In estimating trunk flexion it is important to judge how much of the movement occurs at the spinal joints and how much at the hips.

flexion (Figs 95 and 96). Some patients can almost reach their toes, despite a stiff back, simply by flexing unusually far at the hips. (Normally the hamstrings limit hip flexion to about 90 degrees when the knees are straight) The range may be expressed as a percentage of the normal, or as the distance by which the fingers fail to reach the floor *Extension* Instruct the patient to arch the spine backwards,

symptoms; a history of a jarring strain, a fall, or an unaccustomed lifting job may be important.

Significance of sciatica. If pain radiates into the lower limb its character and exact distribution must be ascertained. Two distinct

TABLE IV

ROUTINE CLINICAL EXAMINATION IN SUSPECTED DISORDERS OF THE BACK

1. Local examination of the back, with neurological survey of the lower limbs

(Patient standing)	*Costo-vertebral joints*
Inspection	Range indicated by chest expansion
Bone contours and alignment Soft-tissue contours Colour and texture of skin Scars or sinuses	*Sacro-iliac joints* (Impracticable to assess range) ? Pain on movement imparted by lateral compression of pelvis
Palpation Skin temperature Bone contours Soft-tissue contours Local tenderness	*(Patient recumbent)* **Palpation of iliac fossae** Examine specifically for abscess
Movements *Spinal joints* Flexion Extension Lateral flexion Rotation ? Pain on movement ? Muscle spasm	**Neurological state of lower limbs** Straight leg raising test Muscular system Sensory system Reflexes

2. Examination of potential extrinsic sources of back pain and sciatica

This is important if a satisfactory explanation for the symptoms is not found on local examination. The investigation should include 1) the abdomen, 2) the pelvis, including rectal examination, and 3) the lower limbs.

3. General examination

General survey of other parts of the body. The local symptoms may be only one manifestation of a widespread disease.

types of sciatica can be recognised. If the pain is severe and radiates in a well defined course, and especially if it is accompanied by motor, sensory, or reflex impairment, it suggests mechanical interference with nerve fibres of the lumbar or sacral plexus. On the other hand, if it

interference with one or more of the roots of the sciatic nerve. The pain is easily explained. Even a normal sciatic nerve is tautened by straight leg raising, though not to the point of causing pain. If a root of the nerve is already stretched or anchored, as by a protruded piece of an intervertebral disc or a tumour, the further tautening entailed in lifting the limb is sufficient to cause pain.

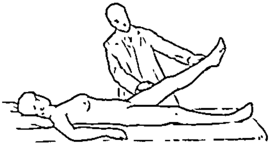

FIG. 98

The straight leg raising test, an important part of the neurological examination of the lower limbs.

Muscular system. Examine the muscles for wasting, hypertrophy, and fibrillation. Note the tone and test the power of each muscle group, comparing it with its counterpart in the opposite limb. Circumferential measurement is a reliable method of comparing the bulk of the calf muscles, the girth being measured at the widest part or "equator" (Fig. 99). Circumferential measurement of the thighs, on

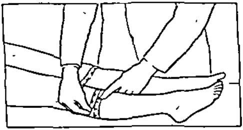

FIG. 99

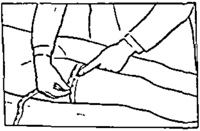

FIG. 100

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the other hand, tends to be inaccurate, and may be misleading, on account of the conical shape of the thigh (Fig. 100). Often a more accurate assessment of the relative volume of the two thighs is obtained from inspection and palpation. If the thighs are measured the girth should be taken on each side at an equal distance above the knee—five or six inches above the upper margin of the patella is usually a convenient level.

Sensory system. Examine the patient's sensibility to touch and pin-prick. When indicated, test also the sensibility to deep stimuli, joint position, vibration, and heat and cold.

Reflexes. Compare on the two sides the knee jerk (mainly L.4) and

looking up at the ceiling. Judge the range and express approximately as a percentage of the normal. *Lateral flexion* : Instruct the patient to slide each hand in turn down the lateral side of the corresponding thigh. Observe the range. *Rotation* : With the feet fixed, the patient rotates the shoulders towards each side in turn. Note the range of spinal rotation as distinct from that which occurs at the knees and hips.

Related joints. *The costo-vertebral joints* : The mobility of the costo-vertebral joints is judged from the range of chest expansion. The normal difference in chest girth at full inspiration and full expiration is about three inches. A marked reduction of chest expansion is of particular significance when ankylosing spondylitis is suspected. *The sacro-iliac joints* : It is not practicable to measure the range of sacro-iliac movement. But the joints should be moved passively to determine whether pain is produced, as it will be in arthritic conditions of the joints. A simple method is to grip each iliac crest and compress the pelvis strongly from side to side.

Palpation of the Iliac Fossae and Groins

Palpation of the iliac fossae and groins is an essential step in the examination of the back. Its specific purpose is to determine whether or not there is a soft-tissue thickening or abscess. It should be remembered that the "psoas" abscess originating from a tuberculous lesion of the lumbar spine first becomes palpable deep in the iliac fossa. Such an abscess is felt most easily by pressing the flat palmar surface of the hand and fingers against the flat inner aspect of the iliac bone. To do this the surgeon must stand at the side of the couch corresponding to the side being examined—that is, he must stand on the right of the patient to examine the right iliac fossa and on the left to examine the left iliac fossa (Fig. 97).

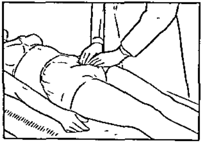

FIG. 97

Palpating the iliac fossae for abscess. This is an essential step in the routine examination of the spine.

Neurological Examination of the Lower Limbs

Disorders of the back are so frequently accompanied by radiating pain, paraesthesiae, or other manifestations in the lower limb that a neurological survey should be carried out as a routine.

Straight leg raising test. Holding the knee straight, lift each lower limb in turn to determine the range of pain-free movement (normal = 90 degrees ; often more in women) (Fig. 98). When associated with clearly defined sciatica (and in the absence of gross disease of the hip), marked impairment of straight leg raising denotes mechanical

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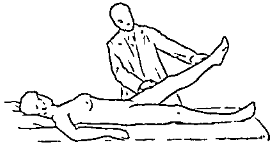

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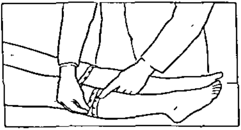
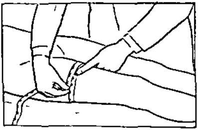


FIG. 99



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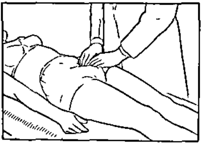

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CLASSIFICATION OF DISORDERS OF THE TRUNK AND SPINE

CONGENITAL ABNORMALITIES

Lumbar and sacral variations
Hemivertebra
Spina bifida
Spondylolysis

DEFORMITIES

Scoliosis
Kyphosis
Lordosis

INFECTIONS OF BONE

Tuberculosis of the thoracic or lumbar spine
Pyogenic infection of the thoracic or lumbar spine

ARTHRITIS OF THE SPINAL JOINTS

Rheumatoid arthritis
Osteoarthritis
Ankylosing spondylitis
Charcot's osteoarthropathy

OSTEOCHONDRITIS

Scheuermann's vertebral osteochondritis
Calvé's vertebral osteochondritis

MECHANICAL DERANGEMENTS

Prolapsed lumbar intervertebral disc
Spondylolysis
Spondylolisthesis

TUMOURS

Tumours in relation to the spinal column, spinal cord, or nerve roots
Other tumours of the trunk

CHRONIC STRAINS

Chronic lower lumbar ligamentous strain
Coccydynia

the ankle jerk (mainly S.1). It is important to note not only the presence or absence of the response, but also any difference of intensity (Figs. 101 and 102). Test the plantar reflex.

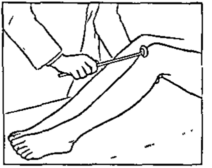


FIG 101

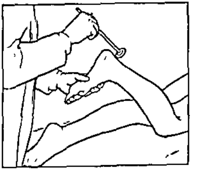


FIG. 102

Figure 101—The patellar reflex is dependent mainly on L.4 nerve
Figure 102—In testing the calcaneal reflex (mainly S.1 nerve), slight inequalities between the two sides can best be detected if the patient lies prone, with the knee flexed 45 degrees and the ankle at 90 degrees

Radiographic Examination

If the complaint is clearly localised to the thoracic spine, antero-posterior and lateral radiographs of that area alone will usually suffice. If the lumbar spine is the part complained of, radiographs should include not only antero-posterior and lateral views of the lumbar spine, but also at least one view of the sacro-iliac joints, pelvis, and hip joints. In cases of doubt additional projections may be required. Oblique projections—from half right and half left—are essential for the proper study of the sacro-iliac joints and the posterior intervertebral (facet) joints of the lumbar region. For doubtful lesions of the vertebral bodies tomographs are often helpful. If a spinal tumour is suspected, myelography is required

Extrinsic Sources of Back Pain and Sciatica

The back offers many pitfalls in diagnosis. Sometimes there are no local symptoms to indicate that the spine is the seat of the disorder, pain being referred entirely to the buttock or to the lower limb. Thus patients frequently complain only of pain " in the hip " or " in the leg " when the true source of the trouble is the lumbar spine. Conversely, the symptoms may suggest a spinal lesion when in fact they arise from an affection of the abdomen, pelvis, or lower limb Finally, it should always be remembered that back symptoms may be no more than a local manifestation of a generalised skeletal disease.

Thus the investigation of back or sciatic symptoms must extend further than a study of the spine itself, it must often include an examination of the abdomen, pelvis, and lower limbs, and a general survey of the rest of the body.

(meningo-myelocele), or there is simply a cystic herniation of the dura (meningocele). These severe forms of spina bifida fall within the province of the neurosurgeon, and they will not be considered further here.

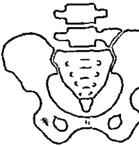


FIG. 103

FIG. 104

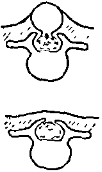


FIG. 105

Three congenital anomalies of the spine. Figure 103—Hypertrophied transverse process forming false joint with ilium. Figure 104—Hemivertebra, an occasional cause of scoliosis. Figure 105—Two examples of spina bifida. In both, the neural arch is deficient posteriorly. In the upper drawing the overlying soft tissues are also deficient and the spinal theca bulges backwards to form a meningocele. The lower drawing shows the much commoner and less severe defect, with the skin and soft tissues intact—spina bifida occulta.

In the minor varieties of spina bifida the skin is intact, only the bony canal being deficient (spina bifida occulta). The defect is often of no clinical significance, but it may be associated with neurological disturbance in the lower limbs and pelvic viscera, probably from adherence of the cauda equina to the bony spinal canal or from associated congenital defects in the spinal cord. The commonest effects are pes cavus, talipes equino-varus, and incontinence of urine.

Treatment. In mild cases treatment is not required. Foot deformities are treated according to established principles (Chapter X). When neurological disturbance is severe, exploration, with freeing of tethered nerve roots, is worth considering.

SPONDYLOLYSIS

In spondylolysis there is a lack of bony continuity in the neural arch of a lower lumbar vertebra (usually the fifth). It predisposes to spondylolisthesis. Although it is usually regarded as a congenital abnormality its pathogenesis is by no means certain. It is described on page 177.

MISCELLANEOUS

Fibrositis
Senile osteoporosis

DISORDERS OF THE SACRO-ILIAC JOINTS

Tuberculosis of a sacro-iliac joint
Pyogenic infection of a sacro-iliac joint
Ankylosing spondylitis
Sacro-iliac ligamentous strain

LUMBAR AND SACRAL VARIATIONS

Minor variations of the bony anatomy are common, especially in the lumbar and sacral regions. They are of little practical importance. They include: deficient or rudimentary lowest ribs; incomplete or complete incorporation of the fifth lumbar vertebral body in the sacrum (sacralisation of the fifth lumbar vertebra); persistence of the first sacral segment as a separate vertebra (lumbarisation of first sacral vertebra); and over-development of the fifth lumbar transverse process on one or both sides, with, in marked cases, a false joint between the hypertrophied process and the ilium (this false joint is sometimes a source of pain) (Fig. 103).

HEMIVERTEBRA

In this anomaly a vertebra is formed in one lateral half only. The defect may occur at any level The body of the half-vertebra is wedge-shaped, and the spine is angled laterally at the site of the defect (Fig. 104) This anomaly is a rare cause of scoliosis.

SPINA BIFIDA

In spina bifida the walls of the vertebral canal fail to meet posteriorly during development. The spinous process and a variable amount of the posterior vertebral arch are deficient (Fig. 105). The anomaly is generally confined to the sacrum or lowest lumbar vertebrae In the worst cases the meninges and soft tissues are also deficient posteriorly, and cerebrospinal fluid discharges from the exposed thecal space. In less complete examples nerve tissue bulges backwards within the intact meninges

(meningo-myelocele), or there is simply a cystic herniation of the dura (meningocele). These severe forms of spina bifida fall within the province of the neurosurgeon, and they will not be considered further here.

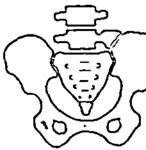


FIG. 103

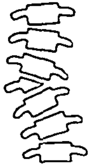
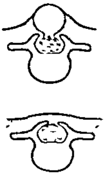


FIG. 104



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SCOLIOSIS

The term scoliosis denotes lateral curvature of the spine. The deformity may be "structural," implying a permanent change in the bones or soft tissues, or it may be no more than a temporary disturbance produced by reflex or postural activity of the spinal muscles. Four types can be recognised: 1) primary or idiopathic structural scoliosis, a well defined group of unknown cause arising in children, 2) secondary structural scoliosis, a miscellaneous group in which the curvature is secondary to a demonstrable underlying disorder; 3) sciatic scoliosis, a temporary deformity; and 4) compensatory scoliosis.

IDIOPATHIC STRUCTURAL SCOLIOSIS

Idiopathic scoliosis is the most important type of structural scoliosis. It begins in childhood or adolescence and tends to increase progressively until the cessation of skeletal growth. It sometimes leads to severe and ugly deformity, especially when the thoracic region is the part affected.

Cause. No satisfactory explanation of its origin has been found.

Pathology. Any section of the thoraco-lumbar spine may be affected. There is a primary curve, with secondary compensatory curves. The pattern of curve and its natural evolution are fairly constant for each site, and the following types are recognised: lumbar scoliosis, thoraco-lumbar scoliosis, thoracic scoliosis (Fig. 106).

Clinical features. The onset may be at any time from infancy to adolescence—often between the ages of 10 and 12. In children deformity is usually the only symptom. Pain is occasionally a feature in adults with long-standing deformity.

Course and prognosis. The outlook depends upon the age at onset and upon the site of the primary curve. The ultimate visible deformity tends to be worst in thoracic scoliosis and least in lumbar scoliosis. The curvature tends to increase until the end of the period of spinal growth, but not thereafter. In general, therefore, the earlier the onset the worse the prognosis.

Treatment. The first essential is to assess the prognosis from a consideration of the age at onset and site of curve. When the

FIG 106
Idiopathic scoliosis in an adolescent girl The main curve is in the thoracic

prognosis is good (for instance, in most cases of lumbar scoliosis) expectant treatment, with review every six months, is all that is required. When the prognosis is poor (as in thoracic scoliosis arising early) active treatment is advised. At present the most satisfactory method is to correct the deformity so far as possible by a hinged plaster jacket controlled by turnbuckles, and subsequently to fuse the whole length of the primary curve by bone grafting to maintain the correction.

It must be emphasised that it is impossible to prevent the increase of a progressive deformity by external appliances or by exercises ; and it is impossible to correct longstanding deformity in adults by any method.

SECONDARY STRUCTURAL SCOLIOSIS

In this group the spinal curvature is secondary to a demonstrable underlying abnormality

Causes. The three commonest underlying causes are congenital abnormalities (especially hemivertebra), poliomyelitis with residual weakness of the spinal muscles, and spinal neurofibroma.

Pathology. In *congenital hemivertebra* there is a sharp angulation at the site of the anomaly, with compensatory curves above and below (Fig 104) Scoliosis following *poliomyelitis* is explained by unequal muscle pull on the two sides. The mechanism of scoliosis complicating spinal *neurofibroma* (often part of a multiple neurofibromatosis) is unknown ; in this type the deformity may be very severe.

Clinical features. In most cases the visible deformity is the only symptom. The age of onset, site, nature, and severity of the curve vary with the underlying cause.

In exceptional cases of severe long-standing scoliosis the sharp angulation of the spinal cord over the apex of the deformity may lead to interference with its function, with consequent neurological manifestations.

Treatment. In most cases treatment is along the lines suggested for idiopathic scoliosis.

SCIATIC SCOLIOSIS

Sciatic scoliosis is a temporary deformity produced by the protective action of muscles in certain painful conditions of the spine.

Cause. The underlying cause is nearly always a prolapsed intervertebral disc impinging upon a lumbar or sacral nerve root. Rarely a tumour or abscess in a similar position may be responsible.

Pathology. The curve is in the lumbar region. The abnormal posture is assumed involuntarily in an attempt to reduce as far as possible the painful pressure upon the nerve root.

Clinical features. The predominant feature is severe back pain or sciatica, aggravated by movements of the spine (see prolapsed intervertebral disc, p 172). The onset is usually sudden. The scoliosis is poorly compensated, so the trunk may be tilted over markedly to one side (Fig 123, p 174). The curvature is not associated with rotation of the vertebrae.

Treatment. The treatment is that of the underlying condition.

COMPENSATORY SCOLIOSIS

Lumbar scoliosis is seen as a compensatory device when the pelvis is tilted laterally—as, for instance, when the lower limbs are unequal *in length*, or *when there is a fixed abduction or* adduction deformity at one hip joint In such a case it is only by curving the lumbar spine through an angle equal to the pelvic tilt that the trunk can be held vertical Usually there is no intrinsic abnormality of the spine itself, and the scoliosis disappears automatically when the pelvic tilt is corrected In cases of many years' duration, however, the lumbar scoliosis may become fixed by adaptive shortening of the tissues on the concave side.

KYPHOSIS

Kyphosis is the general term used to define excessive posterior curvature of the spinal column. The deformity may take the form of a long rounded curve ("round back"), or there may be a localised sharp posterior angulation ("hump back").

In the thoracic region there is normally a considerable posterior curvature: thoracic kyphosis exists only if this curve is excessive. In the cervical and lumbar regions there is normally an anterior curvature: any reversal of this constitutes cervical or lumbar kyphosis.

Causes. Kyphosis is a manifestation of an underlying disorder of the spine. The causes are numerous. The following are the most important: 1) tuberculosis of the spinal column; 2) unreduced vertebral compression fracture; 3) Scheuermann's osteochondritis; 4) senile osteoporosis; and 5) tumour of the spinal column (especially metastatic carcinoma).

Treatment. The treatment is that of the underlying condition.

LORDOSIS

Lordosis is the opposite of kyphosis. The term denotes excessive anterior curvature of the spinal column. In practice, lordosis is seen only in the lumbar region, where a slight anterior curve is normal. Strictly, the term lordosis should be used only when this normal curve is exaggerated.

Causes. Spinal disorders nearly always cause kyphosis or scoliosis rather than lordosis. In many cases lordosis is simply a postural deformity, predisposed to by lax muscles and heavy abdomen. Sometimes it is compensatory, balancing a kyphotic deformity above or below, or a fixed flexion deformity of a hip.

TUBERCULOSIS OF THE THORACIC OR LUMBAR SPINE

(Tuberculous spondylitis, Pott's disease)

Tuberculosis of thoracic or lumbar vertebral bodies is one of the commonest forms of skeletal tuberculosis.

Cause. It is caused by infection with the tubercle bacillus, human or bovine.

Pathology. The infection begins at the anterior margin of a vertebral body, near the intervertebral disc (Fig. 107) The disc

itself is always involved at an early stage. The extent of the destruction varies widely from case to case. Commonly there is complete destruction of one intervertebral disc with partial destruction of the two adjacent vertebrae, most marked anteriorly (Fig. 108). But the changes may extend over several spinal segments; or, on the other hand, they may be confined to a single intervertebral disc, without evident bone involvement (Fig. 109).

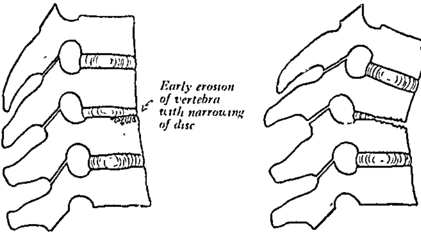

FIG. 107 FIG. 108

Tuberculosis of the spine. The infection begins anteriorly near an intervertebral disc, which is soon destroyed (Fig. 107). It may spread to adjacent vertebrae, which collapse in front, with consequent angular kyphosis (Fig. 108).

Anterior collapse of the affected vertebrae leads to an angular kyphosis. Abscess formation is usual. In the thoracic region the pus collects around the spinal column, forming a fusiform paraspinal abscess (Fig. 111); or it may track towards the surface between ribs. From the lower thoracic or lumbar region pus tracks downwards behind the fascial sheath of the psoas muscle and generally bursts into the compartment behind the iliacus fascia to form a palpable abscess in the iliac fossa (psoas abscess). An abscess occasionally points posteriorly or in the thigh.

Secondary effects. An abscess or mass of granulation tissue encroaching upon the spinal canal may interfere with the spinal cord or cauda equina. In cases of long-standing severe kyphosis the spinal cord is occasionally damaged by the bony ridge at the site of deformity.

Clinical features. The disease is commonest in young adults. One or more of the following symptoms may be present: 1) pain in the back; 2) stiffness of the back; 3) visible deformity of the back; 4) localised swelling (abscess); 5) weakness of legs or visceral dysfunction (involvement of spinal cord). *On examination*, the patient often looks ill. There may be visible or palpable

FIG 109

FIG. 110

Figure 109—Early tuberculous lesion causing a narrowed lumbar disc space in a young adult. Narrowing of a disc space without osteophytic spurring of the vertebral margins always suggests infection. Figure 110—Tuberculous erosion of three lumbar vertebrae, with anterior collapse. One disc is destroyed and the adjacent ones are narrowed.

angular kyphosis. There is local tenderness on firm palpation or percussion over the affected vertebrae. All spinal movements are restricted and when they are attempted the spinal muscles go into protective spasm. An abscess may be detected over the thoracic wall or in the flank, iliac fossa, or upper thigh. Signs of spinal cord compression or of a cauda equina lesion ("Pott's paraplegia") may be present. Tuberculous lesions are often manifest elsewhere. *Radiographic examination:* The earliest signs are narrowing of an intervertebral space (Fig. 109) and local vertebral osteoporosis,

Later, there is usually destruction of bone at the anterior margin of one or more of the vertebral bodies, leading to anterior collapse and wedge-shaped deformity of the affected vertebrae (Figs. 110 and 111). An abscess shadow is nearly always visible: in the thoracic region it is seen as a fusiform paraspinal shadow (Fig. 111); in the lumbar region it is indicated by lateral bulging of the

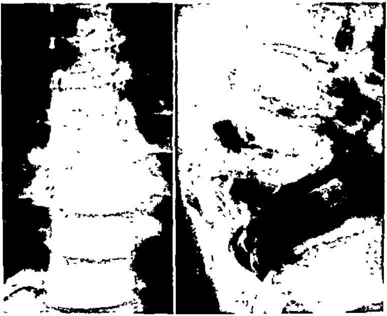

FIG. 111

Tuberculosis of the thoracic spine in a child Note in the antero-posterior view the typical fusiform appearance of a paraspinal abscess, an almost constant feature of thoracic spinal tuberculosis. In the lateral view two vertebral bodies are seen partly collapsed into a wedge-shaped mass, the disc between them has been destroyed

psoas outline, usually on one side. In the healing stage the bone outline in the area of destruction becomes sharper, and normal density is regained Abscesses may become calcified *Investigations:* The erythrocyte sedimentation rate is raised. The Mantoux test is positive Tubercle bacilli can sometimes be isolated from aspirated pus

Diagnosis. The condition has to be distinguished: 1) from other causes of back pain with localised bony destruction or kyphosis (non-specific pyogenic spondylitis; ankylosing spondylitis; spinal

tumours; osteochondritis; old compression fracture), and 2) from other causes of dysfunction of the spinal cord or cauda equina (prolapsed intervertebral disc; spinal tumour). Important diagnostic features are: a history of contact with tuberculosis, or of previous tuberculous infection; restriction of *all* spinal movements by protective muscle spasm; abscess formation; the characteristic radiographic changes (including destruction of an intervertebral disc, which is not usual in cases of tumour and not marked in osteochondritis); and the raised erythrocyte sedimentation rate.

Complications. These include: 1) chronic discharging sinus; 2) interference with the spinal cord or cauda equina, causing weakness or paralysis in the lower limbs with or without sensory impairment or disturbance of bladder and bowel function ("Pott's paraplegia"); and 3) tuberculous infection of other organs, such as meninges, kidneys, or lungs.

Prognosis. In most cases the ultimate outlook is good. In a few there is severe permanent kyphosis. There is a small but definite risk of death from spread of the infection to other organs, or from the effects of paraplegia.

Treatment. *Constitutional treatment* is by rest in a country orthopaedic hospital, good diet, and chemotherapy in the form of streptomycin, para-amino-salicylic acid, and isonicotinic acid hydrazide given together in a six-months' course if well tolerated. *Local treatment* is by rest for the spine on a plaster bed or a frame until the disease is quiescent. Abscesses are aspirated or drained as required. Signs of quiescence are: good general health, diminishing blood sedimentation rate, and arrest of the destructive process with hardening of the bony outlines as seen radiographically. When the disease is quiescent the patient is allowed up with the protection of a plaster jacket or spinal brace, which is worn for about a year.

Indications for operation. Operation is advisable: 1) for relief of paraplegia from spinal cord compression if progressive improvement in the neurological condition is not observed during four weeks' complete rest; 2) to drain a paraspinal abscess that is believed to be retarding healing; and 3) to fuse the affected region of the spine if it is judged to be unstable when the disease has become quiescent.

PYOGENIC INFECTION OF THE THORACIC OR LUMBAR SPINE

(Pyogenic spondylitis ; osteomyelitis of the spine)

Pyogenic infection of the spine is rather uncommon. It has often been confused with tuberculous spondylitis, which it resembles clinically and radiographically.

Cause. It is caused by infection with the streptococcus, staphylococcus, pneumococcus, or occasionally with other bacteria such as the typhoid bacillus.

Pathology. Organisms reach the spinal column through the general blood stream (from a septic focus elsewhere), through the spinal venous plexus (from a focus in the pelvis), or possibly through lymphatic channels (from a neighbouring focus). As in tuberculous spondylitis, there is destruction of bone and intervertebral disc, with or without abscess formation. The spinal cord may be damaged by pressure, or by thrombosis of its vessels.

Clinical features. The onset is usually acute or subacute, and is accompanied by pyrexia. In other respects the clinical features are like those of tuberculous spondylitis (p. 157), the predominant findings being local pain and restriction of all movements by muscle spasm. A suppurative process elsewhere in the body is usually present or has recently subsided. *Radiographs* show local osteoporosis or erosion of bone, diminution of disc space, and sometimes sub-ligamentous new bone formation. After healing, spontaneous bony fusion of affected vertebrae is often observed. *Investigations :* The erythrocyte sedimentation rate is raised. Polymorphonuclear leucocytosis is to be expected.

Diagnosis. The condition has to be distinguished mainly from tuberculous spondylitis, which it may resemble closely. The relatively acute onset, history of pre-existing infection or septic focus, pyrexia, leucocytosis, and identification of the causal organism in pus, are the main diagnostic criteria.

Prognosis. If the infection is overcome, complete healing of the spinal lesion—often with bony fusion of affected vertebrae—is usual

Treatment. This is best undertaken in a country orthopaedic hospital. Appropriate chemotherapy is given. The spine is immobilised on a plaster bed or a frame until healing occurs Abscesses may require drainage, especially if the spinal cord is threatened.

RHEUMATOID ARTHRITIS OF THE SPINAL JOINTS

(General description of rheumatoid arthritis, p. 34.)

The term "rheumatoid spine" is often used loosely to denote ankylosing spondylitis, especially in America. Such terminology is inaccurate and leads to confusion. In point of fact, rheumatoid arthritis of the spinal joints is quite distinct from ankylosing spondylitis.

Pathology. The joint changes are like those of rheumatoid arthritis elsewhere. Unlike ankylosing spondylitis, which always begins in the sacro-iliac joints and creeps upwards, rheumatoid changes in the spinal joints quite commonly begin in the cervical region.

Clinical features. The changes in the spinal joints usually form only part of a widespread rheumatoid polyarthritis The spinal manifestations consist in aching pain of a rather diffuse type, with impairment of spinal movements. The symptoms develop insidiously, without preceding injury. Examination of the limbs will usually reveal typical rheumatoid changes in several joints. *Radiographs* are not distinctive, but in cases of considerable duration there is osteoporosis of the vertebrae with thinning of the intervertebral disc spaces. These changes never progress to bony ankylosis like the changes of ankylosing spondylitis. *Investigations* : The erythrocyte sedimentation rate is increased while the disease is active.

Treatment. This is along the lines recommended for rheumatoid arthritis of other joints (p. 36)

OSTEOARTHRITIS OF THE THORACIC AND LUMBAR SPINE

(Spondylosis, spondylarthrosis)

Osteoarthritis of the thoracic or lumbar intervertebral joints is found very commonly in those used to heavy work, but it is not necessarily accompanied by symptoms.

Cause. Predisposing factors are : 1) previous injury to the spinal joints ; 2) previous disease involving the joints (for example, Scheuermann's osteochondritis) ; and 3) intervertebral disc lesions. In other cases the degenerative changes are simply a manifestation of increasing age.

Pathology. The changes affect the central intervertebral

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Pathology. Organisms reach the spinal column through the general blood stream (from a septic focus elsewhere), through the spinal venous plexus (from a focus in the pelvis), or possibly through lymphatic channels (from a neighbouring focus). As in tuberculous spondylitis, there is destruction of bone and intervertebral disc, with or without abscess formation. The spinal cord may be damaged by pressure, or by thrombosis of its vessels

Clinical features. The onset is usually acute or subacute, and is accompanied by pyrexia. In other respects the clinical features are like those of tuberculous spondylitis (p. 157), the predominant findings being local pain and restriction of all movements by muscle spasm. A suppurative process elsewhere in the body is usually present or has recently subsided. *Radiographs* show local osteoporosis or erosion of bone, diminution of disc space, and sometimes sub-ligamentous new bone formation. After healing, spontaneous bony fusion of affected vertebrae is often observed. *Investigations:* The erythrocyte sedimentation rate is raised. Polymorphonuclear leucocytosis is to be expected.

Diagnosis. The condition has to be distinguished mainly from tuberculous spondylitis, which it may resemble closely. The relatively acute onset, history of pre-existing infection or septic focus, pyrexia, leucocytosis, and identification of the causal organism in pus, are the main diagnostic criteria

Prognosis. If the infection is overcome, complete healing of the spinal lesion—often with bony fusion of affected vertebrae—is usual

Treatment. This is best undertaken in a country orthopaedic hospital. Appropriate chemotherapy is given. The spine is immobilised on a plaster bed or a frame until healing occurs. Abscesses may require drainage, especially if the spinal cord is threatened

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Osteoarthritis of the thoracic or lumbar intervertebral joints is found very commonly in those used to heavy work, but it is not necessarily accompanied by symptoms.

Cause. Predisposing factors are : 1) previous injury to the spinal joints ; 2) previous disease involving the joints (for example, Scheuermann's osteochondritis) ; and 3) intervertebral disc lesions. In other cases the degenerative changes are simply a manifestation of increasing age.

Pathology. The changes affect the central intervertebral

(body-to-body) joints and the posterior intervertebral (facet) joints. One or several segments may be affected. In the central joints, which are affected first, there is degeneration with consequent narrowing of the intervertebral disc, and hypertrophy of bone at the joint margins leads to the formation of osteophytes (Fig. 112). In the posterior intervertebral joints the changes are those of osteoarthritis in any diarthrodial joint—namely, attrition of the articular cartilage and osteophyte formation (lipping) at the joint margins. These changes in the facet joints are probably the more important from a clinical point of view. *Secondary effects :* Rarely, osteophytes encroach upon an intervertebral foramen sufficiently to interfere with the function of the issuing nerve. Thinning of the articular cartilage of the posterior intervertebral (facet) joints reduces the stability of the affected segment and predisposes to one type of spondylolisthesis (p. 178).

Clinical features. Spinal osteoarthritis can exist in quite marked degree without causing symptoms. But there is often a complaint of aching pain in the affected area, worse on activity. Interference with a nerve in a narrowed intervertebral foramen leads to radiating pain in the distribution of the affected nerve (girdle pain or sciatica according to the level affected). The symptoms vary in severity. There is generally a tendency to periodic sharp exacerbations lasting a few weeks or months, with intervals of relative freedom; such exacerbations are possibly explained by unremembered strains of the affected joints *On examination* in the quiet phase, the objective findings are slight Spinal flexion is moderately restricted, but other movements are little if at all impaired, and there is no muscle spasm If there is interference with a sciatic nerve root straight leg raising on the affected side is likely to be restricted Apart from this, objective neurological signs are exceptional During an acute exacerbation

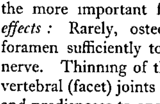


FIG 112

Osteoarthritis of the lumbar spine The notable features are narrowing of the intervertebral disc and "lipping" of the adjacent margins of the vertebral bodies (See also Figure 117)

of the symptoms the positive physical signs are correspondi more pronounced. *Radiographic examination:* The changes most obvious in the central (body-to-body) intervertebral joi which show narrowing of the intervertebral space and osteopl formation (lipping) at the joint margins (Fig. 112). Later posterior intervertebral (facet) joints also show changes: the narrowing of the joint space with sharpening of the margin the facets. These changes are seen clearly only in obl projections.

Diagnosis. Osteoarthritis has to be distinguished from o causes of back pain and from other causes of radiating n pain (girdle pain or sciatica) (Fig. 124, p. 176). It should remembered that the demonstration of osteoarthritic change the radiographs does not necessarily mean that the arthrit the cause of the patient's symptoms, for spinal osteoarthrit often painless. The diagnosis is always presumptive rather proved, and it is justified only when other possible causes been excluded by careful consideration of the history, cli examination, and radiographs.

Treatment. This depends upon the severity of the disab In mild cases treatment is unnecessary. In osteoarthritis of thoracic spine the symptoms are seldom severe, and if treat is required a course of active spinal exercises to strengthen muscles is usually sufficient. In lumbar osteoarthritis moderate disability a well fitted surgical corset will usually a adequate relief. If the pain from a localised lesion is bad en to cause serious hardship operative fusion of the affected segm of the spine may be required.

ANKYLOSING SPONDYLITIS

In ankylosing spondylitis there is chronic inflamma progressing slowly to bony ankylosis, of the joints of the s column and occasionally of the major limb joints.

Cause. This is unknown. The condition is distinct rheumatoid arthritis, although it is sometimes loosely te "rheumatoid spine."

Pathology. The disease always begins in the sacro-iliac jo whence it usually extends upwards to involve the lumbar, thor and often the cervical spine. Occasionally the hips or shou

are also affected. The articular cartilage, synovium, and ligaments show chronic inflammatory changes and eventually they become ossified. After several years the inflammatory process "burns itself out."

FIG 113

Ankylosing spondylitis Characteristic fluffiness with loss of sharp outline of both the sacro-iliac joints Later, these joints undergo spontaneous bony fusion, and similar changes may creep upwards, sometimes affecting the whole of the spinal column

Clinical features. With few exceptions the disease is confined to men, and it nearly always begins between the ages of 20 and 35. *The early symptoms are pain in the lower back and increasing* stiffness. Later, the pain migrates upwards Diffuse radiating pain down one or both lower limbs is also common. *On examination* the predominant finding is marked limitation of all movements in the affected area of the spine ("poker back"). When the thoracic region is involved chest expansion is markedly reduced, from ankylosis of the costo-vertebral joints In a few cases the hips or shoulders are affected, with pain and limitation of movement. *Radiographic examination* · In the early stages there is

"fuzziness," with loss of clear joint outline, in both the sacro-iliac joints (Fig. 113). Later, the sacro-iliac joints are completely obliterated and, if the disease progresses, the intervertebral joints in the lumbar, thoracic, and sometimes even the cervical region

FIG. 114

Ossification of the anterior longitudinal ligament in ankylosing spondylitis.

FIG. 115

Rigid flexion deformity of spine complicating old ankylosing spondylitis. This complication should never be allowed to occur. It can be prevented simply by ensuring that the patient sleeps on his back upon a flat hard mattress, with only a single thin pillow for the head.

undergo bony ankylosis (Fig. 114). *Investigations:* The erythrocyte sedimentation rate is raised while the disease is active.

Diagnosis. Ankylosing spondylitis has to be distinguished from other causes of back pain and sciatica (Fig. 124). The marked limitation of spinal movement, reduced chest expansion, typical radiographic features, and raised erythrocyte sedimentation rate are diagnostic.

Course and complications. The disease usually ceases to progress after five or ten years, leaving permanent stiffness, the extent of which varies widely from case to case. Some patients become bedridden. Complications include gross flexion deformity of the spine (Fig. 115), intercurrent respiratory infections, and iridocyclitis, which in severe cases may lead to blindness.
Treatment. Deep x-ray therapy prevents or retards the progress of the disease, and relieves pain. Active exercises are advisable, to make the most of such movement as remains. The patient is encouraged to sleep flat upon his back on a straight mattress to prevent flexion deformity of the spine. If severe flexion deformity occurs through neglect of this precaution correction by osteotomy of the spine at the lumbar level is worth considering.

CHARCOT'S OSTEOARTHROPATHY OF THE SPINE

(General description of Charcot's osteoarthropathy, p. 47.)

Charcot's disease in the spine is a complication of tabes dorsalis. It affects the lower lumbar or lumbo-sacral region.
Pathology. It can be regarded as a grossly exaggerated form of osteoarthritis; that is to say, there is advanced degeneration of joint surfaces (not only of cartilage but also of underlying bone), with coincident massive hypertrophy of bone at the joint margins. The ultimate configuration of the affected segments is often grotesque. The cauda equina and the issuing nerves are liable to injury from bony displacement or from the pressure of a bony outgrowth
Clinical features. Moderate pain or discomfort in the affected region of the spine is fairly common despite the relative insensitivity to deep pain that is a feature of tabes Movements of the spine are moderately restricted. Other manifestations of tabes dorsalis will be present Sometimes there are coexisting "Charcot" changes in one or more of the joints of the lower limbs. *Radiographs* show marked degenerative destruction of the affected intervertebral joints, much sclerosis of bone, and bony outgrowths from the joint margins.
Treatment. A close-fitting rigid spinal support should be prescribed.

SCHEUERMANN'S VERTEBRAL OSTEOCHONDRITIS

(Adolescent vertebral osteochondritis; adolescent kyphosis)

(General description of osteochondritis, p. 77.)

Scheuermann's disease is usually regarded as osteochondritis of the epiphysial plates of the vertebral bodies. In some respects, however, it behaves differently from typical osteochondritis at other sites, and the precise nature of the disease requires further elucidation.

Cause. The cause is unknown.

Pathology. Characteristically, Scheuermann's disease affects several of the thoracic vertebrae simultaneously. Occasionally the changes are confined to a single vertebra, and this localised form of the disease is probably as frequent in the lumbar as in the thoracic region.

The vertebral bodies ossify mainly from three centres—a primary centre for the middle of the body, and secondary centres for the upper and lower surfaces. The primary centre appears soon after birth, and the secondary centres (known as the epiphysial plates or ring epiphyses) at about the time of puberty. In Scheuermann's disease there is a disturbance of the normal development of these secondary epiphysial plates. They seem to become soft and plastic, and tend to collapse under the stress of weight-bearing. Because of the normal forward curve of the thoracic spine the line of weight-bearing passes in front of the central axis of the column. Consequently the greatest deformation of the epiphyses occurs at their anterior margins, with the result that the affected bodies become wedge-shaped from before backwards (Fig. 116). The deformity predisposes to the later development of osteoarthritis (Fig. 117).

Clinical features. The patient is usually 13 to 16 years old. In the active stage there is pain in the thoracic spine, with " round " back. After some months the pain subsides, leaving a slight rounded kyphosis. In later life there may be renewed pain from the development of osteoarthritis. *On examination*, there is a slight or moderate rounded kyphosis in the thoracic region. In the active stage there is tenderness on firm palpation over the affected vertebrae. *Radiographic examination :* In the active stage of the disease the affected vertebral bodies show deep notched

defects at their anterior corners, and the corresponding parts of the epiphysial plates are irregular in shape, size, and density (Fig. 116). The disc spaces are slightly narrowed but never totally destroyed. After healing, there is slight antero-posterior wedging of the affected vertebral bodies. Years later, osteoarthritic lipping of the anterior vertebral margins is observed (Fig 117).

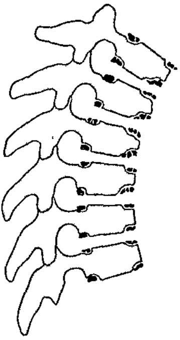


Fig 116

Fig 117

Figure 116—Scheuermann's osteochondritis of the thoracic spine in the active stage (diagrammatic) The ring epiphyses at the upper and lower surfaces of the vertebral bodies are soft and fragmented. They are squashed by the superimposed weight of the trunk, especially in front Figure 117—The late effect of Scheuermann's osteochondritis Wedging of several thoracic vertebral bodies with consequent rounded kyphosis and secondary osteoarthritis

Diagnosis. In its characteristic form the affection *is easily diagnosed from the history, clinical appearance, and radiographs.* If localised to a single vertebra it may easily be confused with tuberculous spondylitis Radiologically, the chief points of distinction are that in osteochondritis the margins of the notched defect in the vertebral body tend to be sclerotic rather than osteoporotic, and a paraspinal abscess shadow (almost a constant feature of thoracic spinal tuberculosis) is never seen Furthermore, the erythrocyte sedimentation rate is not raised in osteochondritis,

Course and prognosis. The affection is self-limiting, the active stage lasting about two years. If the epiphyses become deformed, permanent wedging of the affected vertebrae, with consequent slight or moderate kyphosis, remains. Osteoarthritis often supervenes in later life but it is of little clinical significance.

Treatment. Theoretically the spine should be rested in the anatomical position until the affected epiphyses reharden, a programme that entails lying recumbent in a plaster bed for up to two years. This is seldom practicable for educational and economic reasons, and it is usual to adopt a compromise. If the initial symptoms are severe the child should rest in a plaster bed for three to six months. This allows the pain to subside. Thereafter a spinal brace is worn for a year, and active exercises to strengthen the posterior spinal muscles are encouraged. In milder cases rest in a plaster bed is unnecessary, and a spinal brace with exercises, or even exercises alone, will suffice.

CALVÉ'S VERTEBRAL OSTEOCHONDRITIS

(Vertebra plana)

Whereas Scheuermann's osteochondritis affects the vertebral ring epiphyses, in Calvé's disease it is the central bony nucleus of a vertebral body that is affected. Moreover, unlike the usual form of Scheuermann's disease, which affects several vertebrae, Calvé's disease is confined to a single vertebra. It is much less common than Scheuermann's disease.

Cause. This is unknown.

Pathology. The condition has generally been regarded as analogous to osteochondritis of other developing bony nuclei (p. 77). The bony nucleus of one of the vertebral bodies, usually in the thoracic region, becomes soft and is condensed into a thin wafer. With the natural evolution of the disease there is some regeneration of the flattened bony nucleus, but it is never restored to its full depth. The intervertebral discs above and below are unaffected.

Clinical features. The affection is confined to children of from 2 to 10 years of age. The complaint is of pain, usually in the thoracic region of the spine. *On examination*, there may be a slight localised kyphosis. Percussion of the spinal column reveals

defects at their anterior corners, and the corresponding parts of the epiphysial plates are irregular in shape, size, and density (Fig. 116). The disc spaces are slightly narrowed but never totally destroyed. After healing, there is slight antero-posterior wedging of the affected vertebral bodies. Years later, osteoarthritic lipping of the anterior vertebral margins is observed (Fig. 117).

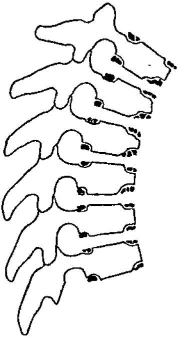


FIG. 116

FIG 117

Figure 116—Scheuermann's osteochondritis of the thoracic spine in the active stage (diagrammatic) The ring epiphyses at the upper and lower surfaces of the vertebral bodies are soft and fragmented They are squashed by the superimposed weight of the trunk, especially in front. Figure 117—The late effect of Scheuermann's osteochondritis Wedging of several thoracic vertebral bodies with consequent rounded kyphosis and secondary osteoarthritis

Diagnosis. In its characteristic form the affection is easily diagnosed from the history, clinical appearance, and radiographs If localised to a single vertebra it may easily be confused with tuberculous spondylitis Radiologically, the chief points of distinction are that in osteochondritis the margins of the notched defect in the vertebral body tend to be sclerotic rather than osteoporotic, and a paraspinal abscess shadow (almost a constant feature of thoracic spinal tuberculosis) is never seen. Furthermore, the erythrocyte sedimentation rate is not raised in osteochondritis.

protrusion herniates through the posterior ligament and may impinge upon an issuing nerve to cause sciatic pain. The nerve affected is that which leaves the spinal canal at the interspace next

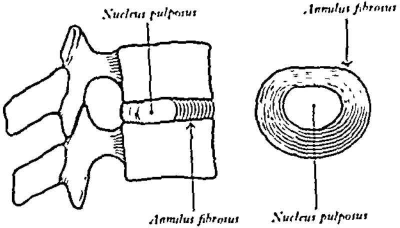


FIG. 119

A normal intervertebral disc seen in sagittal section (left) and in horizontal section (right).

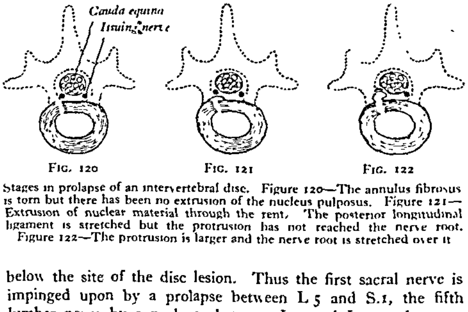


FIG. 120 FIG. 121 FIG. 122

Stages in prolapse of an intervertebral disc. Figure 120—The annulus fibrosus is torn but there has been no extrusion of the nucleus pulposus. Figure 121—Extrusion of nuclear material through the rent. The posterior longitudinal ligament is stretched but the protrusion has not reached the nerve root. Figure 122—The protrusion is larger and the nerve root is stretched over it

below the site of the disc lesion. Thus the first sacral nerve is impinged upon by a prolapse between L.5 and S.1, the fifth lumbar nerve by a prolapse between L.4 and L.5, and so on. Natural healing is probably by shrinkage and fibrosis of the extruded disc material rather than by its spontaneous reposition

deep tenderness in the affected region. Movements of the spine as a whole are impaired but little, if at all. *Radiographs* show the characteristic extreme flattening of the affected vertebral body, which appears strikingly increased in density (Fig. 118). It has been likened to a coin seen end on. The intervertebral disc spaces above and below are of normal depth ; or they may even appear to be increased in depth, for the cartilage of the vertebral body shrinks less than its bony nucleus.

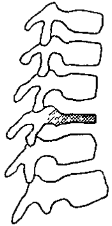


FIG. 118
Calvé's disease of a vertebral body. The bony nucleus has shrunk into a thin dense "wafer." The adjacent intervertebral discs are intact.

Diagnosis. The radiographic appearance is distinctive. It serves to differentiate the condition from tuberculous disease, which always destroys the intervertebral disc.

Treatment. Calvé's disease is non-progressive, and in practice treatment is required only for as long as the symptoms last. Ideally the spine should be relieved of compression forces, *and the only effective way of achieving this is* to nurse the patient recumbent upon a spinal frame or plaster bed. In most cases the patient can safely resume an active life without external support after three to six months.

PROLAPSED LUMBAR INTERVERTEBRAL DISC

Herniation of part of the nucleus pulposus of a lumbar intervertebral disc through the annulus fibrosus accounts for a large proportion of the cases of back pain and sciatica seen in orthopaedic practice.

Cause. Prolapse of a disc is often precipitated by injury, but it is believed that spontaneous age-degeneration of the disc is an important predisposing factor

Pathology. The discs between L.5 and S.1 and between L.4 and L.5 are those most often affected. Part of the gelatinous nucleus pulposus protrudes through a rent in the annulus fibrosus at its weakest part, which is postero-lateral (Figs. 119-122.) If it is small, the protrusion bulges the pain-sensitive posterior longitudinal ligament, causing pain in the back. If it is large, the

limb and is scarcely perceptible in the back. The severity of the pain varies greatly from case to case, and its exact distribution depends upon the level of the disc prolapse; for instance, in the relatively uncommon cases of high lumbar or mid-lumbar prolapse the pain radiates towards the groin and the front of the thigh rather than to the back of the thigh and leg.

Radiographic examination. In a case of acute prolapsed disc the radiographs show no abnormality, and the purpose of radiography is simply to exclude other causes of back pain and sciatica. It is only when a disc has been deranged for many months or years that appreciable narrowing of the disc space and lipping of the joint margins (denoting secondary osteoarthritis) are observed.

Investigations. Lumbar puncture reveals either normal cerebrospinal fluid or, commonly, a slight increase of protein content.

Correlation of pathology with clinical features. The initial injury or strain marks the time when the annulus fibrosus is torn or damaged. The nucleus pulposus is very gelatinous and an interval elapses before it becomes extruded. Bulging of the extruded material beneath the posterior longitudinal ligament corresponds to the stage of acute back pain. Herniation through the ligament with impingement against the adjacent nerve is responsible for the radiating limb pain.

Diagnosis. Prolapsed intervertebral disc must be differentiated from other causes of back pain and sciatica (Fig. 124). The conditions with which it is most likely to be confused are: tuberculosis of the spine or sacro-iliac joints; tumours of the spine or pelvis; spondylolisthesis; ankylosing spondylitis; and osteoarthritis of the spine. Diagnosis rests upon the recognition of the characteristic features of disc prolapse and upon the exclusion of other possible causes of the symptoms by careful consideration of the history, clinical examination (including a rectal examination), and radiographs. A dramatically sudden onset is always suggestive of a mechanical derangement and especially of a prolapsed disc. In doubtful cases lumbar puncture, and sometimes myelography, are required.

Treatment. *Non-operative treatment* is successful in relieving the symptoms in at least nineteen out of every twenty cases. The principle is to provide rest for the lumbar spine. This is preferably secured by a plaster corset (Fig. 125), which allows the

within the disc. *Secondary effects :* Progressive degeneration of the disc leads, after months or years, to osteoarthritis, with ultimate involvement of the posterior intervertebral (facet) joints as well as the central (body-to-body) joints.

Clinical features. In a typical case the clinical picture is clearly defined. The patient is aged between 18 and 60. A few hours or days after jarring or straining the back he is seized, while twisting, stooping, or coughing, with agonising pain in the lumbar region. He is unable to move. The acute pain gradually lessens in severity, but after a few days a radiating pain is felt in one or other buttock and down the back or side of the thigh to the calf and foot. Tingling or numbness is felt in the calf or foot. The pain is aggravated by coughing or sneezing. *On examination* the patient with a fully developed acute attack stands either with a lumbar scoliosis (sciatic scoliosis) (Fig. 123) or with the normal anterior lumbar curve obliterated. Forward flexion is greatly restricted, as also may be extension. Lateral flexion, on the other hand, is usually free and painless—certainly to one side if not to both. Straight leg raising is restricted on the affected side. Careful tests reveal slight muscle wasting or weakness in the distribution of the affected nerve, and the corresponding tendon jerk (knee jerk in L.3-L.4 lesions; ankle jerk in L.5-S.1 lesions) is impaired or absent.

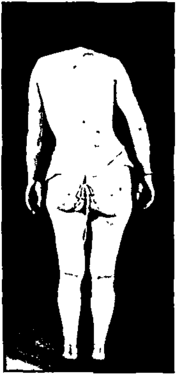


FIG. 123

Sciatic scoliosis A temporary tilt to one or other side is a common feature of severe prolapsed lumbar disc The posture is adopted involuntarily to relieve the pressure upon the nerve at the site of the prolapse

Variations. Atypical cases are common. Thus a definite history of injury or strain is often lacking. The pain may begin gradually rather than suddenly The symptoms may be confined to the back and never radiate to the lower limb (acute lumbago). On the other hand, the pain is sometimes felt predominantly in the

limb and is scarcely perceptible in the back. The severity of the pain varies greatly from case to case, and its exact distribution depends upon the level of the disc prolapse; for instance, in the relatively uncommon cases of high lumbar or mid-lumbar prolapse the pain radiates towards the groin and the front of the thigh rather than to the back of the thigh and leg.

Radiographic examination. In a case of acute prolapsed disc the radiographs show no abnormality, and the purpose of radiography is simply to exclude other causes of back pain and sciatica. It is only when a disc has been deranged for many months or years that appreciable narrowing of the disc space and lipping of the joint margins (denoting secondary osteoarthritis) are observed.

Investigations. Lumbar puncture reveals either normal cerebrospinal fluid or, commonly, a slight increase of protein content.

Correlation of pathology with clinical features. The initial injury or strain marks the time when the annulus fibrosus is torn or damaged. The nucleus pulposus is very gelatinous and an interval elapses before it becomes extruded. Bulging of the extruded material beneath the posterior longitudinal ligament corresponds to the stage of acute back pain. Herniation through the ligament with impingement against the adjacent nerve is responsible for the radiating limb pain

Diagnosis. Prolapsed intervertebral disc must be differentiated from other causes of back pain and sciatica (Fig. 124). The conditions with which it is most likely to be confused are: tuberculosis of the spine or sacro-iliac joints; tumours of the spine or pelvis; spondylolisthesis; ankylosing spondylitis; and osteoarthritis of the spine. Diagnosis rests upon the recognition of the characteristic features of disc prolapse and upon the exclusion of other possible causes of the symptoms by careful consideration of the history, clinical examination (including a rectal examination), and radiographs. A dramatically sudden onset is always suggestive of a mechanical derangement and especially of a prolapsed disc. In doubtful cases lumbar puncture, and sometimes myelography, are required.

Treatment. *Non-operative treatment* is successful in relieving the symptoms in at least nineteen out of every twenty cases. The principle is to provide rest for the lumbar spine. This is preferably secured by a plaster corset (Fig. 125), which allows the

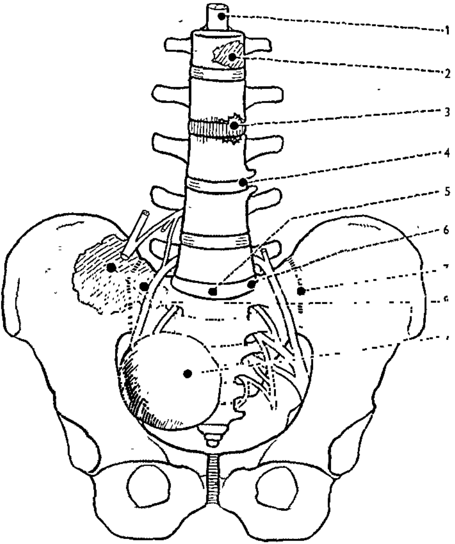


Fig 124

Nine causes of sciatica. All must be considered in the differential diagnosis of prolapsed intervertebral disc 1. Tumour of cord or cauda equina 2 Tumour of spinal column 3 Tuberculosis of spine 4. Osteoarthritis. 5 Spondylolisthesis 6 Prolapsed intervertebral disc. 7 Ankylosing spondylitis 8 Tumour of ilium or sacrum. 9. Intra-pelvic mass

patient to be up and about; but rest in bed, with or without leg traction, is a commonly used alternative. In either case rest for the spine must be continued for six to twelve weeks, according to progress. Thereafter a surgical corset (Fig. 126) should be

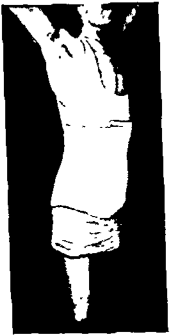


FIG. 125
Plaster corset used in the conservative treatment of prolapsed lumbar intervertebral disc.

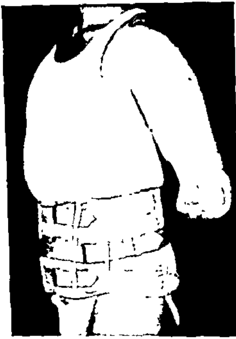


FIG. 126
Steel-reinforced surgical corset used for mild cases of prolapsed intervertebral disc and for certain types of chronic low back pain.

worn for several months if minor symptoms persist. *Operative treatment :* Excision of the displaced disc material is indicated in the following circumstances: 1) When the sciatic pain is so excruciating from the beginning that it prevents sleep and leads to deterioration of the general health; 2) when severe neurological disturbance suggests massive prolapse with compression of the cauda equina; 3) when severe sciatic pain is unrelieved by twelve weeks' efficient conservative treatment.

SPONDYLOLYSIS

In spondylolysis there is a defect in the neural arch of the fifth (rarely the fourth) lumbar vertebra. There is loss of bony

continuity between the superior and the inferior articular processes, the deficiency being bridged by fibrous tissue (Fig. 127). If this stretches or gives way, the consequent vertebral displacement constitutes one variety of *spondylolisthesis* (see below).

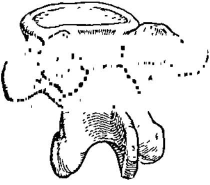


FIG 127
Site of the defect in spondylolysis. There is lack of bony continuity at the isthmus of the neural arch (pars interarticularis) on each side.

Though the defect has hitherto been regarded as congenital it is now believed by some that it may be caused by injury.

Clinically, spondylolysis (the defect without displacement) is often symptomless, but it is believed that it is sometimes a cause of deep lumbar back pain. *Radiographically, the defect is shown clearly only in oblique projections.*

Treatment. This is often unnecessary. Aching may be relieved by a surgical corset. Exceptionally, local fusion of the spine is justified.

SPONDYLOLISTHESIS

(Lumbar spondylolisthesis)

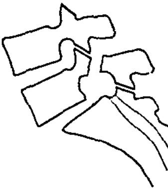
Spondylolisthesis is the term applied to spontaneous forward displacement of a lumbar vertebral body upon the segment next below it.

Cause. The two predisposing factors are: 1) spondylolysis (possibly a congenital defect), and 2) instability of the posterior (facet) articulations, either from osteoarthritis or (rarely) from congenital malformation of the articular processes.

Pathology. In the normal spine forward displacement of a vertebral body is prevented by engagement of its articular processes with those of the segment next below it. In spondylolisthesis there is a failure of this check mechanism, and the attachments of the intervertebral disc alone are not strong enough to hold the vertebral bodies in alignment

In the best recognised type ("true" spondylolisthesis) a defect in the neural arch of a vertebra (Fig. 127) allows separation of its two halves. The body, with the pedicles and superior

articular processes (and the whole of the spinal column above it), slips forwards, leaving behind the laminae and inferior articular processes (Fig. 128). The fifth lumbar is the vertebra usually affected, the fourth occasionally. Displacement may increase slowly month by month or year by year, and it sometimes reaches a severe degree. There may be minor irritation of one of the



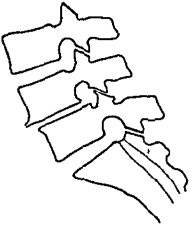


FIG. 128 FIG. 129

Figure 128—Spondylolisthesis due to defect of the neural arch (diagrammatic). The body and superior articular processes have slipped forwards, leaving the spinous process and inferior articular [illegible] cally [illegible] 29— [illegible] the cartilage of the posterior intervertebral (facet) joints has permitted slight forward displacement of the fourth lumbar vertebra on the fifth. The condition may occur at any level in the lumbar spine.

issuing nerves, with consequent sciatica; but despite severe bony displacement gross interference with the nerves of the cauda equina is exceptional in this type of spondylolisthesis.

In the second type of spondylolisthesis (inaccurately termed "pseudo-spondylolisthesis") displacement of one vertebra upon another is permitted by instability of the posterior (facet) articulations. It may occur at any level in the lumbar spine. The commonest cause of instability is osteoarthritis of the posterior (facet) joints, with attrition of the articular cartilage that is essential to a snug fit of the joint surfaces (Fig. 129). Less often, the facet joints are congenitally unstable, from malformation of the articular processes. In the usual osteoarthritic type the displacement is always slight, and neurological disturbance is unusual.

When the articular processes are congenitally deficient, however, there may be severe displacement with trapping of the cauda equina.

Clinical features. Spondylolisthesis is often symptomless When symptoms occur, they take the form of chronic backache, with or without sciatica. The back pain is worse on standing. *On examination* there is often a visible or palpable "step" above the sacral crest, due to the forward displacement of the spinal column ; but this is obvious only when the displacement is severe. Spinal movements are restricted only slightly, if at all. *Abdomen :* When displacement is severe the spinal column is projected forwards and the lumbar vertebral bodies may be palpable through the abdomen. *Lower limbs :* Minor irritation of a sciatic root is often evidenced by impairment of straight leg raising ; but severe neurological disturbance is seldom observed except in the rare cases in which congenital malformation of the articular processes allows dislocation of the whole vertebra complete with its neural arch. *Radiographs* show the displacement. Oblique views will demonstrate whether or not there is a defect of the neural arch.

Diagnosis. Spondylolisthesis is distinguished from other causes of back pain and sciatica by the radiographs.

Treatment. When spondylolisthesis is symptomless treatment is not required. *Non-operative treatment :* Moderate symptoms are often adequately relieved by a well fitted surgical corset, and this should be tried before operation is considered. *Operation :* This is justified only when the disability (from back pain or neurological disturbance) is severe. The operation entails the release of stretched or compressed nerves, followed by fusion of the affected segments of the spinal column.

TUMOURS OF THE TRUNK AND SPINE

These tumours are best considered in two categories: 1) Tumours that affect the spinal column or its contents—spinal cord or nerve roots ; and 2) other tumours of the trunk.

TUMOURS IN RELATION TO THE SPINAL COLUMN, SPINAL CORD, OR NERVE ROOTS

Classification and pathology. *Tumours of the spinal cord or meninges :* These include meningioma, intradural neurofibroma and, rarely, glioma *Tumours of nerves :* The important example

is neurofibroma, which may be single or multiple. If it arises from a nerve in an intervertebral foramen it may grow inwards to compress the spinal cord and outwards towards the surface ("dumb-bell" tumour). *Tumours of the bony spinal canal:* Benign tumours of the spinal column are much less common than malignant tumours. They include chondroma, osteoclastoma, and vertebral haemangioma. Malignant tumours may be primary (sarcoma, multiple myeloma, chordoma), but much more frequently they are metastatic tumours derived usually from a carcinoma of the lung, breast, prostate, thyroid, or kidney (hypernephroma).

Clinical features. The effects of these tumours vary according to their site and character. Broadly, the effects may be placed in three groups: 1) compression of the spinal cord; 2) local destruction of the skeleton; 3) interference with peripheral nerves.

Compression of the spinal cord. This may occur with tumours of the spinal cord itself or of its meninges, with tumours of nerves (neurofibroma), or with tumours of the bony spinal column. The clinical manifestations depend upon the exact location of the tumour. Typically, when the spinal cord is slowly compressed, initial root pain (girdle pain in thoracic involvement, lower limb pain in lumbar) is followed by lower motor neurone paresis at the segmental level corresponding with the site of the tumour, and by progressive sensory and upper motor neurone paralysis below the lesion, often with bladder or bowel dysfunction.

FIG. 130
Collapse of a lumbar vertebra caused by a carcinomatous metastasis. Note that the intervertebral discs are not destroyed

Local destruction of the skeleton. The commonest cause is a malignant tumour of the bones of the spinal column—usually a

metastatic carcinoma (Fig. 130). The predominant symptom is pain, which is constant and increases relentlessly in its severity. Frequently there are associated neurological manifestations from involvement of the spinal cord or nerve roots. The local objective signs vary from case to case. Sometimes the tumour is palpable —as, for instance, a sacral tumour. Sometimes deformity, from collapse of the bony structure, is evident clinically ; or there may be marked restriction of spinal movement, with protective muscle spasm.

Interference with peripheral nerves. Peripheral nerves—especially the nerves of the cauda equina—may be involved by tumours of the nerves themselves (neurofibroma), by tumours of the spinal column (benign or malignant), or by tumours in the peripheral course of the nerves (for example, a tumour of a rib, or a tumour arising from or within the pelvis). The clinical features depend upon the particular nerve or nerves affected and upon the extent of the involvement. Typically, there will be constant, progressive, and ultimately severe pain along the course of the affected nerve, with sensory impairment, increasing motor weakness, and depression of reflexes in the distribution of the nerve. Retention of urine is usually a prominent feature of a tumour interfering with the cauda equina.

Radiographic examination. Plain radiographs will usually help in discovering a tumour arising in the bones of the spinal column, or eroding the bone from outside. Myelography is indispensable if a tumour of the spinal cord or cauda equina is suspected. Radiographs of the chest may reveal a primary lung tumour or a metastasis ; and radiographs of the rest of the skeleton may be helpful in the diagnosis of disseminated tumours such as multiple myeloma.

Investigations. The following are of value in appropriate cases : lumbar puncture (for intraspinal tumour) ; urine tests for Bence-Jones proteose (for multiple myeloma or leukaemia) ; blood counts (for leukaemia) ; acid phosphatase estimation (for prostatic metastases) ; and marrow biopsy (for leukaemia, lymphadenoma, multiple myeloma, carcinomatous metastases) Careful search should always be made for a possible primary tumour.

Diagnosis. Tumours in the region of the spinal column are easily mistaken for other disorders that can produce similar effects.

Mainly, they have to be distinguished from three groups of conditions: 1) from other causes of compression of the spinal cord (massive prolapsed intervertebral disc; gross scoliosis or kyphosis); 2) from other causes of local destruction of the spinal column (tuberculous or pyogenic infection; syphilis); and 3) from other causes of peripheral nerve disturbance (tuberculous or pyogenic infection with involvement of nerve roots; prolapsed intervertebral disc; spinal osteoarthritis; spondylolisthesis; herpes zoster; neurological disorders such as disseminated sclerosis).

A history of insidious onset, with relentless increase of symptoms, always suggests the possibility of a tumour. Findings that lend support to the possibility include: history or demonstration of primary tumour; demonstrable metastases elsewhere—for instance, in the lungs; markedly raised protein content of the cerebrospinal fluid (in spinal tumours); and a raised acid phosphatase content of the blood (in prostatic tumours).

Radiographically, an important point of distinction between tumours of the vertebral bodies and destructive infections such as tuberculosis is that, whereas the intervertebral disc is relatively resistant to tumours and is seldom destroyed by them, infective lesions nearly always attack and destroy the disc at an early stage (compare Figs. 110 and 130).

Treatment. This depends upon the character of the individual tumour. In general, the tumour should be excised when excision is practicable. If it is not, recourse must be had to palliative measures appropriate to the tumour. These include deep x-ray therapy, gonadal hormone therapy with or without castration or adrenalectomy (for metastases from breast or prostatic tumours), cortisone (for leukaemia), and analgesic drugs as required.

OTHER TUMOURS OF THE TRUNK

Tumours of the Sternum and Ribs

The sternum and ribs contain abundant red marrow, favourable to the development of blood-borne metastatic tumours. They are also affected commonly in multiple myeloma. Histological examination of the sternal marrow (obtained by sternal puncture) is often of diagnostic importance in suspected metastasising

metastatic carcinoma (Fig. 130). The predominant symptom is pain, which is constant and increases relentlessly in its severity. Frequently there are associated neurological manifestations from involvement of the spinal cord or nerve roots. The local objective signs vary from case to case. Sometimes the tumour is palpable —as, for instance, a sacral tumour. Sometimes deformity, from collapse of the bony structure, is evident clinically; or there may be marked restriction of spinal movement, with protective muscle spasm.

Interference with peripheral nerves. Peripheral nerves—especially the nerves of the cauda equina—may be involved by tumours of the nerves themselves (neurofibroma), by tumours of the spinal column (benign or malignant), or by tumours in the peripheral course of the nerves (for example, a tumour of a rib, or a tumour arising from or within the pelvis). The clinical features depend upon the particular nerve or nerves affected and upon the extent of the involvement. Typically, there will be constant, progressive, and ultimately severe pain along the course of the affected nerve, with sensory impairment, increasing motor weakness, and depression of reflexes in the distribution of the nerve Retention of urine is usually a prominent feature of a tumour interfering with the cauda equina

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Diagnosis. Tumours in the region of the spinal column are easily mistaken for other disorders that can produce similar effects.

Diagnosis. This depends upon the exclusion of demonstrable pathological lesions by careful clinical and radiographic examination. A history of long-continued lumbar backache, with a total lack of clinical or radiological abnormalities, should always suggest this group of affections.

Course and prognosis. Aching often persists for many years despite treatment. Nevertheless in most cases the condition is a source of nagging discomfort rather than a serious handicap to the normal activities of life.

Treatment. Often reassurance alone is required. When treatment is called for, the three methods available are: 1) physiotherapy; 2) external support; and 3) manipulation. In young patients physiotherapy, in the form of active exercises to strengthen the spinal muscles, with or without heat and massage, should be tried for at least two months. If this is ineffective, fitting with a surgical corset is advised. In elderly and flabby patients physiotherapy is seldom of benefit and early resort should be had to a reinforced corset. If pain persists despite these measures manipulation of the lumbar and sacro-iliac joints under anaesthesia is sometimes worth a trial.

COCCYDYNIA

In its widest sense, coccydynia includes any painful condition in the region of the coccyx. In practice, the term is restricted to the clinical entity in which persistent pain continues for many weeks or months after a local injury, despite the absence of demonstrable gross pathology. Eventually it is a self-limiting affection, though it may cause severe discomfort while it lasts.

Cause. Typically, coccydynia develops after an injury—usually a fall onto the "tail." Occasionally, a history of injury is lacking.

Pathology. There are no gross changes. In some cases there is probably a strain of the sacro-coccygeal joint; in others the lesion is thought to be simply a contusion of the periosteum over the lower sacrum or coccyx.

Clinical features. There is pain localised to the sacro-coccygeal area, worse when sitting. In severe cases there is also pain on defaecation. Usually the patient is free from pain when standing or lying *On examination* there is localised tenderness over the sacro-coccygeal region. In some cases the pain can be reproduced

tumours, for the material will often show tumour cells even in the absence of clinically evident metastases.

TUMOURS OF THE SCAPULA

The commonest tumour of the scapula is a chondroma. It grows outwards from the flat body of the bone and is therefore classed as an ecchondroma. It may attain a large size. There is a small risk of malignant change, with the development of a sarcoma. For that reason a chondroma that appears to be enlarging should always be excised with an adequate margin of healthy bone. A large part of the scapula can be removed without causing serious subsequent disability.

TUMOURS OF THE PELVIC GIRDLE

The pelvic bones, like the scapula, are sometimes the seat of a chondroma (ecchondroma). It may reach a large size, and there is some risk of malignant change.

The considerable content of red marrow renders the pelvic bones liable to carcinomatous metastatic deposits, and they are also a favourite site of tumour deposits in multiple myeloma.

CHRONIC LOWER LUMBAR LIGAMENTOUS STRAIN

(Postural back pain)

The terms chronic ligamentous strain and postural back pain are used to cover an ill defined group of affections characterised by persistent backache without demonstrable pathology. These conditions are common—in fact they form a large proportion of the cases of back pain seen in orthopaedic practice.

Cause. It is assumed that the spinal muscles fail in their function of protecting the deep ligaments in maintaining posture. Predisposing causes include childbirth, overweight, general flabbiness of muscle, and debilitating illness.

Pathology. No precise lesion is demonstrable.

Clinical features. The patient is nearly always a woman. She often dates the onset of pain from childbirth, sometimes from an operation, or from a debilitating illness; but equally often the onset is unexplained. The pain is characteristically in the lumbar or lumbo-sacral region. It tends to be worse on activities such as stooping. *On examination* there are no abnormal physical signs. *Radiographs* are normal.

Diagnosis. This depends upon the exclusion of demonstrable pathological lesions by careful clinical and radiographic examination. A history of long-continued lumbar backache, with a total lack of clinical or radiological abnormalities, should always suggest this group of affections.

Course and prognosis. Aching often persists for many years despite treatment. Nevertheless in most cases the condition is a source of nagging discomfort rather than a serious handicap to the normal activities of life.

Treatment. Often reassurance alone is required. When treatment is called for, the three methods available are: 1) physiotherapy; 2) external support; and 3) manipulation. In young patients physiotherapy, in the form of active exercises to strengthen the spinal muscles, with or without heat and massage, should be tried for at least two months. If this is ineffective, fitting with a surgical corset is advised. In elderly and flabby patients physiotherapy is seldom of benefit and early resort should be had to a reinforced corset. If pain persists despite these measures manipulation of the lumbar and sacro-iliac joints under anaesthesia is sometimes worth a trial.

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Cause. Typically, coccydynia develops after an injury—usually a fall onto the " tail." Occasionally, a history of injury is lacking.

Pathology. There are no gross changes. In some cases there is probably a strain of the sacro-coccygeal joint; in others the lesion is thought to be simply a contusion of the periosteum over the lower sacrum or coccyx.

Clinical features. There is pain localised to the sacro-coccygeal area, worse when sitting. In severe cases there is also pain on defaecation. Usually the patient is free from pain when standing or lying. *On examination* there is localised tenderness over the sacro-coccygeal region. In some cases the pain can be reproduced

by moving the coccyx. *Radiographs* show no alteration from the normal.

Diagnosis. It is important to consider alternative causes of pain in this area, especially infections of the sacro-coccygeal joint and tumours of the sacrum or coccyx. The investigation should include rectal examination, and radiographs must always be obtained.

Treatment. Various remedies have been advocated, ranging from short-wave diathermy and injection of local anaesthetic to excision of the coccyx. In fact, however, treatment is not required All that is necessary is to exclude serious organic lesions, and to reassure the patient about the nature of the trouble and the ultimate outcome.

FIBROSITIS

The general subject of fibrositis was discussed on page 108 A loose diagnosis of "fibrositis" is often made in cases of thoracic or lumbar back pain, but in fact it is an uncommon condition—some even deny its existence. In many cases of supposed fibrositis the cause of the pain is more probably a ligamentous strain or an intervertebral disc lesion.

The cause of fibrositis is unknown, and there are no demonstrable pathological changes. So-called fibrositic nodules cannot be identified histologically.

Clinical features. There is aching pain in the posterior spinal muscles of varying intensity and often influenced by climatic changes. *On examination* there is local tenderness on palpation of the affected muscles. Apart from this, no clinical abnormality is found : spinal movements are full and there is no muscle spasm. *Radiographs* show no abnormality. "Fibrositic" pains in other parts of the body commonly coexist.

Treatment. This is by physiotherapy in the form of local heat, with or without massage or exercises.

SENILE OSTEOPOROSIS

Although the symptoms of senile osteoporosis are predominantly in the back, it is strictly a general affection of the skeleton and was described as such on page 91. The spinal features are aching pain, kyphosis, liability to compression fractures of vertebral bodies, and marked rarefaction of the bones of the spinal column.

DISORDERS OF THE SACRO-ILIAC JOINTS

Sacro-iliac lesions are a rather uncommon but nevertheless important cause of back symptoms or of referred pain in the lower limb.

TUBERCULOSIS OF A SACRO-ILIAC JOINT

The patient is a child or young adult. The condition is usually confined to one side, but it may be bilateral. As in tuberculous disease of other joints, there is destruction of articular cartilage and thickening of synovial membrane, often with erosion of bone and abscess formation. *Clinically*, the main feature is pain behind the affected joint and in the iliac fossa or groin. A diffuse, ill localised pain may be referred down the lower limb. Examination shows restriction of lower spinal movements, and side-to-side compression of the pelvis aggravates the pain. An abscess is often palpable posteriorly or in the iliac fossa. *Radiographs* show local osteoporosis, with loss of definition or "fuzziness" of the joint outline.

Treatment is like that for tuberculosis of other joints—namely, constitutional treatment by rest in a country orthopaedic hospital, with systemic streptomycin, para-amino-salicylic acid, and iso-nicotinic acid hydrazide continued for six months; and local treatment by immobilisation of the affected joint (plaster bed or plaster hip spica) until the disease is quiescent. Thereafter some surgeons allow the patient up in a sacro-iliac support, whereas others advise operative fusion (arthrodesis) of the affected joint as a safeguard against recrudescence of the disease. Abscesses should be aspirated or drained.

PYOGENIC INFECTION OF A SACRO-ILIAC JOINT

This is uncommon. Usually it is unilateral. The infection reaches the joint through the blood stream from a pre-existing source such as a boil, a throat infection, or a pneumonic focus. The infection destroys the joint cartilage, with or without suppuration. Bony ankylosis is usual after healing. *Clinically*, the features are like those of tuberculosis of the joint, but the onset and course are more acute. *Radiographically*, there is no apparent abnormality in the earliest stages; later, there are local

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Diagnosis. It is important to consider alternative causes of pain in this area, especially infections of the sacro-coccygeal joint and tumours of the sacrum or coccyx. The investigation should include rectal examination, and radiographs must always be obtained.

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osteoporosis and loss of definition of the joint outline. The polymorphonuclear leucocyte count is increased.

Treatment is best carried out in a country orthopaedic hospital. General treatment is by rest and systemic chemotherapy appropriate to the causative organism. Local treatment is by rest for the joint and drainage of abscesses.

ANKYLOSING SPONDYLITIS

Although ankylosing spondylitis usually affects a larger area of the spine (see p. 165) it always begins in the sacro-iliac joints and is sometimes confined to them. It always affects both joints The initial chronic inflammatory lesion progresses eventually to bony ankylosis of the joints. The patient is nearly always a man of 20 to 35 years. There is pain across the base of the spine, and often also a diffuse referred pain down one or both lower limbs. Examination reveals marked stiffness in the lower part of the spine. There is no abscess formation. *Radiographs* show marked loss of definition or "fuzziness" of both sacro-iliac joints, sometimes with irregular marginal sclerosis: eventually the changes progress to bony ankylosis.

Treatment. The most effective treatment is deep x-ray therapy to the affected joints. In the exceptional cases in which the disease affects women irradiation of the sacro-iliac joints is not justifiable (except by a less effective modified technique) because of the risk of damaging the ovaries.

SACRO-ILIAC LIGAMENTOUS STRAIN

Sacro-iliac strain was formerly a commonplace diagnosis in cases of pain localised predominantly in the upper gluteal region. It is now believed that in the vast majority of such cases the causative condition is an intervertebral disc lesion, the local "sacro-iliac" pain being simply a referred pain originating in the lumbar spine Exceptionally, however, a true sacro-iliac ligamentous strain occurs *Clinically*, the patient is usually an adult woman The symptoms are often noticed first after childbirth. The pain is accurately localised to the sacro-iliac joint, and there is a tendency also to referred diffuse aching in the thigh. The pain is aggravated by twisting the trunk. Examination reveals a

good range of spinal movements with pain only at the extremes. Forcible stress applied to the sacro-iliac joints by firm lateral compression of the pelvis or by passive rotation of the trunk upon the pelvis reproduces the pain. There are no neurological signs. *Radiographs* show no abnormality.

Treatment. Genuine sacro-iliac strain often responds well to manipulation under anaesthesia. If this fails, recourse is usually made to physiotherapy (heat, massage, exercises) or, this failing also, to a surgical corset.

EXTRINSIC DISORDERS SIMULATING SPINAL DISEASE

ABDOMINAL DISORDERS

PEPTIC ULCER

The pain of peptic ulcer is often felt in the back as well as in the epigastrium. Exceptionally, it is felt entirely in the back or beneath the left costal margin, when it may simulate girdle pain referred along a thoracic spinal nerve.

VISCEROPTOSIS

Dragging of pendulous viscera upon the posterior abdominal wall is a contributory cause of backache Flabbiness of the back muscles, with consequent ligamentous strain, is usually present as well. The pain from both conditions can be relieved by fitting a combined abdominal and lumbo-sacral support.

RENAL OR PERIRENAL INFECTIONS

Rarely, a carbuncle of the kidney or a perinephric abscess is confused with a back condition. The usual features are pain in the back and loin, general malaise, pyrexia, and leucocytosis

RENAL CALCULUS

The pain may be felt chiefly in the back, though it is always towards the loin rather than in the midline. The pain is often aggravated by jarring movements—for instance, riding on a shaky bus or stepping off a pavement Haematuria is a frequent symptom, and there is sometimes a history of attacks of colic. The range of spinal movements is normal. Radiographs will reveal the calculus.

BILIARY CALCULUS AND CHOLECYSTITIS

Pain beneath the right costal margin from a gall-bladder lesion is easily confused with girdle pain referred along an intercostal nerve from a spinal condition.

PELVIC DISORDERS

INTRAPELVIC TUMOUR

A tumour or other mass within the pelvis may interfere with the sacral plexus or its branches, causing pain which is generally of sciatic distribution. It may thus simulate sciatica arising from a spinal cause such as a prolapsed intervertebral disc or spinal tumour. A pelvic mass will usually be palpable on rectal or vaginal examination, which should form part of the routine investigation in cases of radiating lower limb pain

GYNAECOLOGICAL DISEASE

The importance of gynaecological disorders as a cause of back pain has been exaggerated. In point of fact, there is no reasonable ground for attributing back symptoms to gynaecological causes unless a gross intrapelvic disorder is demonstrable.

LOWER LIMB DISORDERS

ARTHRITIS OF THE HIP

The pain from arthritis of the hip sometimes simulates sciatic pain referred from a spinal lesion. Characteristically, hip pain is referred from the groin down the front of the thigh towards the knee Irritation of the fourth lumbar nerve root by a spinal lesion causes pain in a somewhat similar distribution. The best safeguard against error is to examine the movements of the hip joint in every case of radiating lower limb pain.

CHAPTER FIVE

The Shoulder Region

THE mechanics of the shoulder are rather complex. The shoulder "joint" in fact comprises three components—the gleno-humeral joint or shoulder joint proper, the acromio-clavicular joint, and the sterno-clavicular joint. The gleno-humeral joint allows a free range of abduction, flexion, and rotation, under the control of the scapulo-humeral muscles. The other two joints together allow 90 degrees' rotation of the scapula upon the thorax and a moderate range of antero-posterior gliding of the scapula, under the control of the cervico-scapular and thoraco-scapular muscles.

Disorders of the shoulder include most varieties of arthritis; but it is notable that osteoarthritis—common in most joints—is rare in the gleno-humeral joint. As if to make up for this, the shoulder exhibits several affections peculiar to itself—notably tears of the musculo-tendinous cuff, the painful arc syndrome, and "frozen" shoulder. Together these form a large proportion of shoulder disabilities.

Pain in the shoulder and arm is notoriously prone to misinterpretation, and special care is required to differentiate intrinsic pain arising in the shoulder from extrinsic pain referred from the cervical spine, the thorax, or the abdomen.

SPECIAL POINTS IN THE INVESTIGATION OF SHOULDER SYMPTOMS

History

Characteristics of shoulder pain. It is important to find out the precise location and distribution of the pain. True shoulder pain is seldom confined to the shoulder itself. Typically, it radiates from a point near the tip of the acromion down the lateral side of the upper arm to about the level of the deltoid insertion. It is unusual for true shoulder pain to extend below the elbow.

Pain arising in the acromio-clavicular joint or sterno-clavicular joint is localised to the joint itself and does not radiate down the limb.

Referred pain in the shoulder region. The pain referred from an irritative lesion of the brachial plexus often extends from the base of the neck, over the top of the shoulder, and thence into the arm. Unlike true shoulder pain, it frequently radiates below the elbow into the forearm or hand, and it may be accompanied by paraesthesiae—often described as "pins and needles" or "a numb feeling."

TABLE V

ROUTINE CLINICAL EXAMINATION IN SUSPECTED DISORDERS OF THE SHOULDER

1 LOCAL EXAMINATION OF THE SHOULDER REGION

Inspection Bone contours and alignment Soft-tissue contours Colour and texture of skin Scars or sinuses	**Power** *Cervico-scapular and thoraco-scapular muscles* (controlling scapular movement)—Elevation of scapula, retraction of scapula, abduction-rotation of scapula.
Palpation Skin temperature Bone contours Soft-tissue contours Local tenderness	*Scapulo-humeral muscles* (controlling gleno-humeral movement)—Abduction, adduction, flexion, extension, lateral rotation, medial rotation
Movements Distinguish between gleno-humeral movement and scapular movement during abduction, flexion, extension, lateral rotation, and medial rotation ? Pain on movement ? Muscle spasm ? Crepitation on movement	**Acromio-clavicular joint** Examine for swelling, increased warmth, tenderness, pain on movement, and stability **Sterno-clavicular joint** Examine for swelling, increased warmth, tenderness, pain on movement, and stability

2 EXAMINATION OF POTENTIAL EXTRINSIC SOURCES OF SHOULDER SYMPTOMS

This is important if a satisfactory explanation for the symptoms is not found on local examination The investigation should include: 1) the neck, with the brachial plexus; 2) the thorax, with special reference to the heart and pleura, and 3) the abdomen, for subdiaphragmatic lesions.

3 GENERAL EXAMINATION

General survey of other parts of the body.

Exposure

The patient must be stripped to the waist. The examination is conducted most easily with the patient standing; alternatively he may sit upon a high stool. For the greater part of the examination the

surgeon stands behind the patient, so that he may observe more easily the position of the scapula.

Steps in Routine Examination

A suggested plan for the routine clinical examination of the shoulder is summarised in Table V.

Movements at the Shoulder

In examining shoulder movements it is important to determine how much of the movement occurs at the gleno-humeral joint and how much is contributed by rotation of the scapula. An accurate distinction between the two types of movement can be made only by grasping the lower half of the scapula so that its movements can be detected (Fig. 131). In the normal shoulder about half the range of abduction occurs at the gleno-humeral joint and half by scapular rotation. Disorders of the shoulder generally cause restriction of gleno-humeral movement rather than of scapular movement.

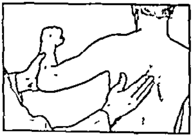

FIG. 131

Examining shoulder abduction. One hand grasps the scapula while the other steadies the elbow. In this way the proportion of the total range contributed by gleno-humeral movement and by scapular rotation can be assessed.

Stand behind the patient. *Abduction* Instruct the patient to try to raise both arms sideways from the body so that the palms of the hands meet above the head. Measure the range, and observe what proportion of the movement takes place at the gleno-humeral joint and how much is contributed by rotation of the scapula upon the thorax. *Flexion:* Instruct the patient to raise the arms forwards towards the vertical. Again observe (by means of the hand upon the scapula) what proportion of the movement occurs at the gleno-humeral joint. *Extension:* Ask the patient to raise the elbows backwards. *Lateral rotation:* The elbows are held in to the sides and are flexed 90 degrees (Fig. 132): the forearms then serve as convenient "pointers" to indicate the angle of rotation (normal range = 80 degrees). *Medial rotation:* Instruct the patient to place the back of his hand in contact with his lumbar region and to carry the elbow forwards (normal range = 110 degrees).

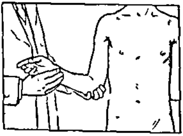

FIG 132

Examining shoulder rotation The elbows are flexed 90 degrees, thereby eliminating forearm rotation The forearms serve as pointers, indicating clearly the range of rotation

Estimation of Muscle Power

In estimating the power of the shoulder muscles two groups must be distinguished: 1) the cervico-scapular and thoraco-scapular muscles; and 2) the scapulo-humeral muscles.

The cervico-scapular and thoraco-scapular muscles. These control movements of the scapula. Estimate the power of each group in turn and compare on the two sides. *Elevators of the scapula* (levator scapulae, upper fibres of trapezius): Instruct the patient to shrug the shoulders against the resistance of the examiner's hands *Retractors of the scapula* (rhomboids and middle fibres of trapezius): Instruct the patient to brace the shoulders back. *Abductor-rotators of the scapula* (serratus anterior, with middle and lower fibres of trapezius): Instruct the patient to push horizontally forwards with the hand against a wall (Fig. 133) or against the resistance of the examiner. If the serratus anterior is weak "winging" of the scapula (backward projection of its vertebral border) will be observed.

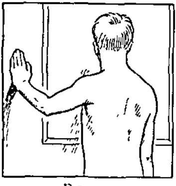

FIG 133
Examination for weakness of the serratus anterior

The scapulo-humeral muscles. These control movements of the gleno-humeral joint. Estimate the power of each muscle group, testing in turn the abductors, adductors, flexors, extensors, lateral rotators, and medial rotators. If the patient has lost the power to initiate active gleno-humeral movement from the dependent position, determine whether he can maintain abduction when the limb has been raised with assistance to 90 degrees. Ability to *sustain* abduction but not to *initiate* it is characteristic of isolated rupture or paralysis of the supraspinatus (Figs 142 and 143, p. 205).

The Acromio-clavicular and Sterno-clavicular Joints

The clavicle can be regarded as a link, jointed at each end, connecting the scapula to the sternum (Fig. 134) Movement of the scapula must occur about a fulcrum at one or both ends of this link In the normal shoulder movement of the scapula, with consequent movement at the acromio-clavicular and sterno-clavicular joints, occurs mainly 1) during elevation of the arm above 90 degrees, and 2) when the shoulders are braced backwards or drawn forwards. To examine the acromio-clavicular and sterno-clavicular joints stand in front of the patient. Examine the joints on each side for deformity, swelling, increase of local temperature, local tenderness, and pain on movement—especially at the extremes of elevation of the arm and backward bracing of the shoulders. Observe whether there is any tendency to subluxation or dislocation of the joint on movement.

Radiographic Examination

The gleno-humeral joint. The routine shoulder film is a plain antero-posterior projection with the limb in the anatomical position. When additional information is required a special supero-inferior

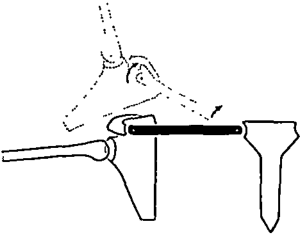

FIG. 134

The mechanics of scapular rotation. The clavicle serves as a link, jointed at each end, by which the scapula is held away from the sternum. Full rotation of the scapula entails movement both at the acromio-clavicular joint and at the sterno-clavicular joint.

projection with the arm abducted 90 degrees (giving a lateral view of the humerus), or stereoscopic films, should be obtained. Further films showing the upper end of the humerus in varying degrees of rotation are sometimes informative.

The acromio-clavicular joint and sterno-clavicular joint. Special projections are used to show each of these joints.

Extrinsic Sources of Shoulder and Arm Pain

In many cases in which the main complaint is of pain in the shoulder or arm there is no local abnormality, the symptoms being referred from a lesion elsewhere. Thus pain over the shoulder is a common symptom in affections of the neck, especially when the brachial plexus or its roots are involved. Shoulder pain is also a feature of irritative lesions in contact with the diaphragm, either in the thorax or in the abdomen. The possibility of such extrinsic lesions must always be considered in

... from extrinsic lesions. The important point is that *intrinsic* lesions of the shoulder always give rise to local physical signs that are readily demonstrable on examination. If the shoulder is clinically normal it is improbable that it is the seat of disease, and attention should be directed towards the possible sources of referred pain.

CLASSIFICATION OF DISORDERS OF THE SHOULDER REGION

DISORDERS OF THE SHOULDER (GLENO-HUMERAL) JOINT

ARTHRITIS

Pyogenic arthritis
Rheumatoid arthritis
Tuberculous arthritis
Osteoarthritis

MECHANICAL DERANGEMENTS

Recurrent dislocation
Complete tear of the tendinous cuff
Rupture of the long tendon of biceps
Painful arc syndrome

MISCELLANEOUS

" Frozen " shoulder
Tenosynovitis of the long biceps tendon.

DISORDERS OF THE ACROMIO-CLAVICULAR JOINT

ARTHRITIS

Osteoarthritis

MECHANICAL DERANGEMENTS

Persistent dislocation and subluxation

DISORDERS OF THE STERNO-CLAVICULAR JOINT

ARTHRITIS

Pyogenic arthritis
Tuberculous arthritis

MECHANICAL DERANGEMENTS

Persistent or recurrent dislocation

DISORDERS OF THE SHOULDER (GLENO-HUMERAL) JOINT

PYOGENIC ARTHRITIS OF THE SHOULDER

(General description of pyogenic arthritis, p. 31.)

Pyogenic arthritis of the shoulder is uncommon. It occurs most often in children, in whom infection may spread to the shoulder from a focus of osteomyelitis in the upper metaphysis of the humerus.

Cause. The staphylococcus, streptococcus, pneumococcus, or gonococcus is the organism usually responsible.

Pathology. The organisms may reach the shoulder 1) through the blood stream, 2) through a penetrating wound, or 3) from an adjacent focus of osteomyelitis. There is an acute or subacute inflammatory reaction, with exudation of turbid fluid or pus within the joint. The outcome varies according to the virulence of the organisms and the resistance of the patient: there may be complete healing with preservation of a normal joint, or there may be partial or total destruction of the joint, with fibrous or bony ankylosis.

Clinical features. The onset is acute or subacute, with pain and loss of function of the shoulder. There is constitutional illness, with pyrexia. *On examination* the shoulder is hot and swollen. All movements are greatly limited by pain, and attempts to move the joint passively provoke protective spasm of the muscles. A primary focus of infection can often be found elsewhere in the body. *Radiographs* show no abnormality in the earliest stage of the disease. Later, if the infection continues, there is diffuse osteoporosis throughout the shoulder area and the cartilage space may be reduced. *Investigations:* The erythrocyte sedimentation rate is raised. There is a polymorphonuclear leucocytosis. Bacteriological examination of aspirated pus usually reveals the identity of the causative organism.

Treatment. *General treatment* is by rest and chemotherapy. Whenever possible the causative organism is identified and the most effective antibiotic drug determined by sensitivity tests: but if the organism cannot be identified empirical chemotherapy by penicillin is justified. *Local treatment:* The joint is rested in

a sling or on a splint. Pus or turbid exudate is removed from the joint by aspiration or, in fulminating suppurative infections, by incision . the appropriate antibiotic solution is instilled in its place. Aspiration and replacement by antibiotics are repeated *daily so long as the effusion re-forms. Rest or immobilisation is* continued until the inflammatory process has resolved. Thereafter active exercises are encouraged.

RHEUMATOID ARTHRITIS OF THE SHOULDER

(General description of rheumatoid arthritis, p. 34.)

The shoulder is affected with moderate frequency, often on both sides. As in other joints, the disease manifests itself as a chronic non-bacterial arthritis, with pain, swelling, and impairment of movement.

Pathology. The synovial membrane of the affected joints is much thickened by chronic inflammatory changes. If the disease progresses, the articular cartilage is slowly softened and eroded. The inflammatory process remains active for months or years, but eventually it subsides, leaving a joint that is more or less damaged.

Clinical features. Typically, several joints are affected either simultaneously or within a short period. The main symptoms are pain and stiffness, worst when activity is resumed after resting. *On examination* the shoulder is abnormally warm, and swollen with synovial thickening. All movements are limited—especially abduction beyond the right angle. The controlling muscles are wasted *Radiographs* show no abnormality at first ; later there is osteoporosis in the region of the joints, with diminution of the cartilage space *Investigations :* The erythrocyte sedimentation rate is increased during the active phase.

Treatment. This is mainly that for rheumatoid arthritis in general, as described on page 36. Exercises are important in maintaining a useful range of movement *Operative treatment :* Excision of the acromion has been recommended for painful limitation of abduction, but the results are uncertain

TUBERCULOUS ARTHRITIS OF THE SHOULDER

(General description of tuberculous arthritis, p 37.)

Tuberculous arthritis of the shoulder is uncommon compared *with disease of the spine, hip, and knee.*

Pathology. The bacilli reach the shoulder through the blood stream. The most striking early changes are in the synovial membrane, which becomes thickened by a chronic inflammatory reaction. Later, if the disease advances, the articular cartilage is destroyed and the underlying bone is eroded. Suppuration, with

FIG. 135

Tuberculous arthritis of the shoulder. Note the loss of articular cartilage and the marked erosion of bone at the joint surfaces

formation of a tuberculous (cold) abscess, is less common in the shoulder than in other tuberculous joints. The term *caries sicca* has been used to describe this " dry " non-suppurative type of disease that characteristically affects the shoulder.

Clinical features. A high proportion of the patients affected are adults who have manifest tuberculosis elsewhere. The general health is impaired. The local symptoms are severe pain in the shoulder and upper arm, stiffness, and marked impairment of function. *On examination* the skin over the shoulder is abnormally warm and the joint is diffusely swollen with " boggy " synovial thickening. In addition, there may be a local fluctuant swelling from an abscess, or a discharging sinus. All movements of the gleno-humeral joint are restricted ; forced movements cause pain and provoke protective muscle spasm. The muscles are wasted.

a sling or on a splint. Pus or turbid exudate is removed from the joint by aspiration or, in fulminating suppurative infections, by incision : the appropriate antibiotic solution is instilled in its place. Aspiration and replacement by antibiotics are repeated daily so long as the effusion re-forms. Rest or immobilisation is continued until the inflammatory process has resolved. Thereafter active exercises are encouraged.

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TUBERCULOUS ARTHRITIS OF THE SHOULDER

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Tuberculous arthritis of the shoulder is uncommon compared with disease of the spine, hip, and knee.

Clinical features. The patient is usually elderly: osteoarthritis is exceptional in the shoulders of younger patients. The main complaint is of pain in the shoulder and down the upper arm. *On examination* there is no increase of local skin temperature and no synovial thickening. But a soft swelling due to effusion of fluid into the joint is common. Movements are restricted. *Radiographs* show narrowing of the cartilage space; the joint outlines are clear-cut and often show some sclerosis; there is "spurring" or "lipping" from osteophyte formation at the joint margins (Fig. 136).

FIG. 136

Osteoarthritis of the shoulder in an old woman. Note loss of articular cartilage, marginal osteophytes or "lipping," and sclerosis at the joint surfaces. Osteoarthritis is uncommon in the shoulder.

Treatment. In many cases no treatment is required once the nature of the affection has been explained. If treatment is called for, conservative measures should usually be relied upon: short-wave diathermy, massage, and gentle exercises are often helpful If there is a large effusion it should be aspirated. In exceptional cases operation is justified: arthrodesis is then the method of choice.

RECURRENT DISLOCATION OF THE SHOULDER

A traumatic dislocation of the shoulder is liable to cause structural changes in the gleno-humeral joint which predispose to repeated dislocations.

Pathology. This is twofold (Figs. 137-139). 1) The capsule is stripped from the anterior margin of the glenoid rim but retains an attachment farther down the neck of the scapula. Thus there is created an intracapsular "pocket" in front of the glenoid margin, into which the humeral head my be displaced (Fig. 138). 2) The articular surface of the humeral head is "dented" posterolaterally (probably by the initial violence) (Figs 138 and 139).

A tuberculous lesion elsewhere is usually discoverable. *Radiographic examination :* In the early stages there is diffuse osteoporosis throughout the gleno-humeral area. Later, there is narrowing of the cartilage space and erosion of the underlying bone (Fig. 135). *Investigations :* The erythrocyte sedimentation rate is increased. The Mantoux test is positive. Aspirated pus may yield tubercle bacilli. Appropriate tests (chest radiograph, sputum and urine examination) may reveal evidence of tuberculosis elsewhere.

Diagnosis. Tuberculosis has to be distinguished from other forms of arthritis, and from other causes of pain and stiffness of the shoulder—especially " frozen " shoulder. Important diagnostic features are the diffuse swelling with increased local temperature, the limitation of all movements, the radiographic appearances, the presence of tuberculous lesions elsewhere, and the identification of the tubercle bacillus in aspirated pus. If the diagnosis is in doubt biopsy should be advised.

Treatment. This is like that for other tuberculous joints. It should be carried out in a country orthopaedic hospital. *Constitutional treatment* is by rest and appropriate antibiotic drugs (streptomycin, para-amino-salicylic acid and isonicotinic acid hydrazide). *Local treatment* is by rest for the joint on a splint or in plaster until the disease is quiescent. Thereafter treatment depends upon the residual state of the joint. If the articular cartilage has been preserved movements are begun ; but if cartilage or bone has been destroyed permanent elimination of the joint by arthrodesis is advised (Fig. 3, p. 18).

OSTEOARTHRITIS OF THE SHOULDER

(General description of osteoarthritis, p. 41.)

Unlike most other joints, the shoulder is very seldom affected by osteoarthritis. When it does occur there is usually a clear predisposing factor, such as previous injury or disease, or senility. The rarity of osteoarthritis of the shoulder is explained by its freedom from pressure stresses.

Pathology. The articular cartilage is worn away The underlying bone becomes eburnated and at the joint margins it hypertrophies to form osteophytes.

The consequent defect in the contour of the articular surface allows the head to subluxate readily over the front of the glenoid when the arm is in lateral rotation and abduction. The dislocation is anterior and it must be emphasised that the humeral head always remains within the capsule.

Clinical features. There is always a history of initial violent dislocation. Thereafter dislocation recurs with trivial violence, characteristically during combined abduction and lateral rotation (for example, in putting on a coat). *On examination* no clinical abnormality is apparent. *Radiographic examination :* Routine radiographs (with the limb in the anatomical position) show no abnormality, but special profile views taken with the arm in 60 to 80 degrees' medial rotation show the characteristic bony defect of the humeral head (Fig. 140).

Treatment. Conservative treatment is not effective. If dislocation recurs frequently operation is justified. The most reliable methods are the Bankart operation, in which the capsule is re-attached to the front of the glenoid margin ; and the Putti-Platt operation, in which the subscapularis tendon is shortened by overlapping or " reefing " in order to limit lateral rotation.

COMPLETE TEAR OF THE TENDINOUS CUFF

(Torn supraspinatus)

It is important to distinguish complete tears of the tendinous cuff [1] from incomplete tears The clinical effects are different. Whereas an incomplete tear is one cause of the " painful arc syndrome," a complete tear impairs seriously the power of shoulder abduction.

Cause. The tendon gives way under a sudden strain, usually imposed by a fall. Age-degeneration of the tendon is a constant predisposing factor.

Pathology. The tear is mainly of the supraspinatus tendon, but it may extend into the adjacent subscapularis or infraspinatus tendons. The tear is close to the insertion of the tendons and usually involves the capsule of the joint, with which the tendons are blended. The edges of the rent retract, leaving a gaping hole

[1] The term *tendinous cuff* denotes the supraspinatus tendon together with the adjoining flat tendons that are blended with it—namely, the infraspinatus behind and the subscapularis in front. They form a cuff over the shoulder that has also been termed, inaccurately, the *rotator cuff*. Distally, the tendons forming the cuff blend with the capsule of the shoulder

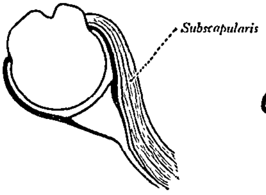

FIG. 137

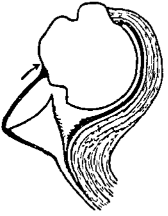

FIG. 138

Horizontal section of left shoulder showing the pathology of recurrent dislocation. Figure 137 shows the normal condition. In Figure 138 the humeral head is shown dislocated forwards. It has stripped the capsule from the margin of the glenoid, creating a pocket in front of the neck of the scapula into which the humeral head is displaced. Note that the humeral head has been "dented" by the sharp glenoid margin, producing the typical defect of the articular surface.

FIG. 139

FIG. 140

Figure 139—Typical defect of articular surface of humeral head, found in most cases of recurrent dislocation of the shoulder. Figure 140—Radiographic appearance. The defect is seen at the upper and outer quadrant of the humeral head.

The consequent defect in the contour of the articular surface allows the head to subluxate readily over the front of the glenoid when the arm is in lateral rotation and abduction. The dislocation is anterior and it must be emphasised that the humeral head always remains within the capsule.

Clinical features. There is always a history of initial violent dislocation. Thereafter dislocation recurs with trivial violence, characteristically during combined abduction and lateral rotation (for example, in putting on a coat). *On examination* no clinical abnormality is apparent. *Radiographic examination :* Routine radiographs (with the limb in the anatomical position) show no abnormality, but special profile views taken with the arm in 60 to 80 degrees' medial rotation show the characteristic bony defect of the humeral head (Fig. 140).

Treatment. Conservative treatment is not effective. If dislocation recurs frequently operation is justified. The most reliable methods are the Bankart operation, in which the capsule is re-attached to the front of the glenoid margin ; and the Putti-Platt operation, in which the subscapularis tendon is shortened by overlapping or " reefing " in order to limit lateral rotation.

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(Torn supraspinatus)

It is important to distinguish complete tears of the tendinous cuff [1] from incomplete tears. The clinical effects are different. Whereas an incomplete tear is one cause of the " painful arc syndrome," a complete tear impairs seriously the power of shoulder abduction.

Cause. The tendon gives way under a sudden strain, usually imposed by a fall. Age-degeneration of the tendon is a constant predisposing factor.

Pathology. The tear is mainly of the supraspinatus tendon, but it may extend into the adjacent subscapularis or infraspinatus tendons. The tear is close to the insertion of the tendons and usually involves the capsule of the joint, with which the tendons are blended. The edges of the rent retract, leaving a gaping hole

[1] The term *tendinous cuff* denotes the supraspinatus tendon together with the adjoining flat tendons that are blended with it—namely, the infraspinatus behind and the subscapularis in front. They form a cuff over the shoulder that has also been termed, inaccurately, the *rotator cuff*. Distally, the tendons forming the cuff blend with the capsule of the shoulder.

which establishes a communication between the shoulder joint and the subacromial bursa (Fig. 141).

Clinical features. The patient is usually a man over 60. After a strain or fall he complains of pain at the tip of the shoulder and

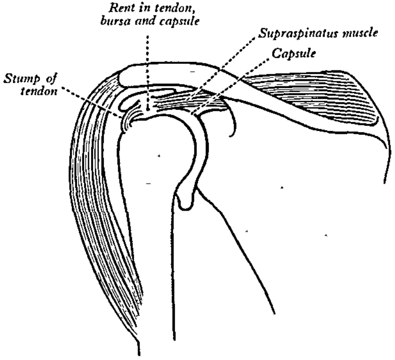

FIG. 141
Tear of supraspinatus shown diagrammatically. Note that the subacromial bursa communicates with the shoulder joint through the rent.

down the upper arm, and of inability to raise the arm. *On examination* there is local tenderness below the margin of the acromion. When the patient attempts to abduct the arm no movement occurs at the gleno-humeral joint but a range of about 45 to 60 degrees of abduction can be achieved, entirely by scapular movement (Fig. 142). There is, however, a full range of passive movement; and if the arm is abducted with assistance beyond 90 degrees the patient can sustain the abduction by deltoid action (Fig. 143). Thus the essential and characteristic feature in cases of "torn supraspinatus" is inability to initiate gleno-humeral abduction. The mechanical explanation is that the early stages of abduction demand the combined action of the deltoid, which

supplies the main motive force, and the supraspinatus, which stabilises the humeral head in the glenoid fossa (like the workman's foot against a ladder that is being raised from the ground).

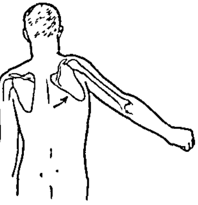

FIG. 142

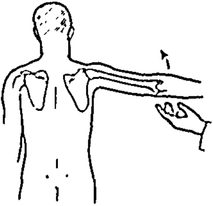

FIG. 143

Complete te[illegible]
abduction fi[illegible]
the deltoid [illegible]
help of the [illegible]
beyond the [illegible]

Diagnosis. Complete tear of the tendinous cuff must be distinguished from other causes of impaired gleno-humeral abduction, especially the painful arc syndrome and paralysis of the abductor muscles (as from poliomyelitis or nerve injury). Inability to initiate gleno-humeral abduction, with power to sustain abduction once the limb has been raised passively, is characteristic of loss of supraspinatus action. In the painful arc syndrome the power of abduction is retained but the movement is painful.

Treatment. In old patients the condition is sometimes best left untreated. Conservative treatment is ineffective. Operative treatment is by suture of the rent; thereafter the arm is rested in abduction on a splint or in plaster for three weeks. The results of operation are not uniformly satisfactory.

RUPTURE OF THE LONG TENDON OF BICEPS

The long tendon of the biceps is one of several tendons in the body that are prone to rupture without violent stress or injury. (Others are the supraspinatus tendon and the tendon of extensor pollicis longus.)

Cause. The tendon will not rupture under ordinary stresses unless it is already weak. The predisposing factor is age-degeneration, probably accelerated by oft-repeated friction and angulation at the point where the tendon enters the bicipital groove of the humerus.

Clinical features. The patient is usually a man past middle age. While lifting or pulling with the arm he feels something give way in the region of the front of the shoulder. There is only moderate discomfort, and often the patient neglects to seek early advice. Later he may notice an unusual bulge of the muscle in front of the arm. *On examination* soon after the rupture, there is slight tenderness over the bicipital groove of the humerus. When the patient contracts the biceps muscle, as in flexing the elbow or supinating the forearm against resistance, the belly of the long head is seen to bunch up into a short round mass like a ball. There is surprisingly little weakness of elbow flexion or of supination

Treatment. When the rupture is diagnosed promptly operation should be undertaken to suture the distal stump of the tendon to the walls of the bicipital groove ; the proximal stump is ignored. In long-established cases the disability is so slight that operation is seldom worth while.

PAINFUL ARC SYNDROME

(Supraspinatus syndrome)

This is a clinical syndrome characterised by pain in the shoulder and upper arm during the mid-range of gleno-humeral abduction, with relative freedom from pain at the extremes of the range The syndrome is common to five distinct shoulder lesions.

Cause. The pain is produced mechanically by nipping of a tender structure between the tuberosity of the humerus and the acromion (or coraco-acromial ligament).

Pathology. Even in the normal shoulder, the clearance between the upper end of the humerus and the acromion process is small in the range of abduction between 45 and 160 degrees If a swollen and tender structure is present beneath the acromion it is liable to get nipped during the arc of movement in which the

clearance is small (Fig. 145), with consequent pain. In the neutral position and in full abduction the clearance is greater and pain is less marked or absent (Figs. 144 and 146).

FIG. 144

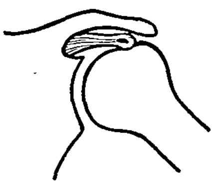

FIG. 145

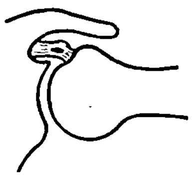

FIG. 146

Mechanical basis of the painful arc syndrome. The black area represents any tender lesion near the supraspinatus insertion. Figure 144—With the arm dependent, the lesion is free from pressure. Figure 145—With the arm in mid-abduction the lesion is nipped between the humerus and the acromion. Figure 146—At full elevation the lesion is again free from pressure.

Five primary lesions can give rise to the syndrome (Fig. 147). 1) Injury of the greater tuberosity (contusion or undisplaced fracture). 2) Minor tear of the supraspinatus tendon. Tearing or strain of a few tendon fibres causes an inflammatory reaction with local swelling, but power is not significantly impaired (compare complete tear of the tendinous cuff). 3) Supraspinatus tendinitis. In this condition there is believed to be an inflammatory

Cause. The tendon will not rupture under ordinary stresses unless it is already weak. The predisposing factor is age-degeneration, probably accelerated by oft-repeated friction and angulation at the point where the tendon enters the bicipital groove of the humerus.

Clinical features. The patient is usually a man past middle age. While lifting or pulling with the arm he feels something give way in the region of the front of the shoulder. There is only moderate discomfort, and often the patient neglects to seek early advice. Later he may notice an unusual bulge of the muscle in front of the arm. *On examination* soon after the rupture, there is slight tenderness over the bicipital groove of the humerus. When the patient contracts the biceps muscle, as in flexing the elbow or supinating the forearm against resistance, the belly of the long head is seen to bunch up into a short round mass like a ball. There is surprisingly little weakness of elbow flexion or of supination.

Treatment. When the rupture is diagnosed promptly operation should be undertaken to suture the distal stump of the tendon to the walls of the bicipital groove; the proximal stump is ignored. In long-established cases the disability is so slight that operation is seldom worth while.

PAINFUL ARC SYNDROME

(Supraspinatus syndrome)

This is a clinical syndrome characterised by pain in the shoulder and upper arm during the mid-range of gleno-humeral abduction, with relative freedom from pain at the extremes of the range. The syndrome is common to five distinct shoulder lesions.

Cause. The pain is produced mechanically by nipping of a tender structure between the tuberosity of the humerus and the acromion (or coraco-acromial ligament).

Pathology. Even in the normal shoulder, the clearance between the upper end of the humerus and the acromion process is small in the range of abduction between 45 and 160 degrees. If a swollen and tender structure is present beneath the acromion it is liable to get nipped during the arc of movement in which the

tendinitis, calcified deposit, or subacromial bursitis. Radiography will confirm or exclude a fracture or a calcified deposit (Fig. 149). (A calcified deposit is distinguished from an avulsed fragment of

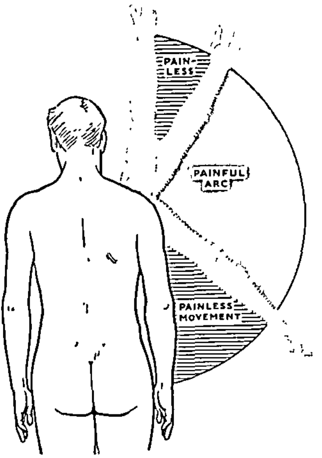

FIG. 148

Painful arc syndrome. The middle arc of abduction is painful whereas the extremes are painless.

bone by the fact that it is homogeneous and does not show the trabeculation characteristic of bone.)

Treatment. In mild cases treatment is often unnecessary. When treatment is called for, the method used should depend upon the primary cause of the syndrome. In most cases non-operative measures are successful. *Contusion or crack fracture of the greater tuberosity*. Reliance should be placed on active use

reaction provoked by degeneration of the tendon fibres. 4) Calcified deposit in the supraspinatus tendon. A white chalky deposit forms within the tendon, and the lesion is surrounded by an inflammatory reaction. 5) Subacromial bursitis. The bursal walls are inflamed and thickened from mechanical irritation.

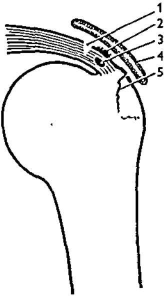

FIG. 147

Five causes of the painful arc syndrome. The clinical features are the same in each. 1 Incomplete tear of supraspinatus 2. Supraspinatus tendinitis. 3. Calcified deposit in supraspinatus 4. Subacromial bursitis. 5. Crack fracture of greater tuberosity

Clinical features. Whatever the primary cause, the clinical syndrome has the same general features, though they vary in degree. With the arm dependent pain is absent or minimal. During abduction of the arm pain begins at about 45 degrees and persists through the arc of movement up to 160 degrees (Fig. 148). Thereafter the pain lessens or disappears. In descent from full elevation pain is again experienced during the middle arc of the range: often the patient will twist or circumduct the arm grotesquely in an effort to get it down with the least pain. The severity of the pain varies from case to case. In extreme cases it is so agonising that the patient is unable to face the ordeal of lifting the arm through the painful arc: a calcified deposit in the supraspinatus tendon is usually responsible for this very acute type.

Diagnosis. Painful arc syndrome is sometimes confused with arthritis of the acromio-clavicular joint, which also causes pain during a certain phase of the abduction arc But in acromio-clavicular arthritis the pain begins later in abduction (not below 90 degrees) and increases rather than diminishes as full elevation is reached.

Differentiation between the five primary causes of the syndrome is aided by the history and by radiography. A history of injury suggests a strain of the supraspinatus tendon or a lesion of the greater tuberosity, whereas a spontaneous onset suggests

tendinitis, calcified deposit, or subacromial bursitis. Radiographs will confirm or exclude a fracture or a calcified deposit (Fig. 149). (A calcified deposit is distinguished from an avulsed fragment of

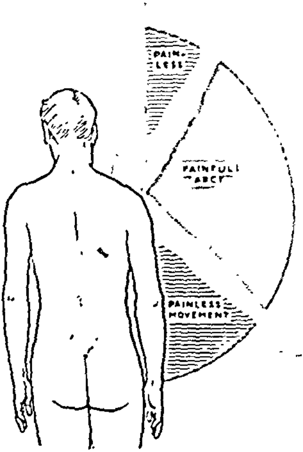

Fig. 148

Painful arc syndrome. The middle arc of abduction is painful whereas the extremes are painless.

bone by the fact that it is homogeneous and does not show the trabeculation characteristic of bone.)

Treatment. In mild cases treatment is often unnecessary. When treatment is called for, the method used should depend upon the primary cause of the syndrome. In most cases non-operative measures are successful. *Contusion or crack fracture of the greater tuberosity:* Reliance should be placed on active use

and mobilising exercises. *Strain of supraspinatus, supraspinatus tendinitis*, and *subacromial bursitis :* Most of these cases respond to short-wave diathermy and mobilising exercises. *Calcified deposit in the supraspinatus tendon :* In cases of moderate severity treatment is by rest in a sling and short-wave diathermy, with mobilising exercises when the pain begins to subside. If the pain is of agonising intensity, as it sometimes is in these cases, immediate relief can be gained by removing the toothpaste-like deposit through an aspiration needle or through an incision into the tendon.

FIG 149
Calcified deposit in the supraspinatus tendon Note that the texture of the deposit is homogeneous It is thereby distinguished from an avulsed fragment of bone, which would show trabeculation

In cases of painful arc syndrome in which troublesome symptoms persist despite a full trial of efficient conservative treatment the acromion should be excised to prevent the possibility of further nipping of inflamed tissue between it and the upper end of the humerus.

"FROZEN" SHOULDER

(Adhesive capsulitis , periarthritis)

" Frozen " shoulder is an ill-understood affection of the glenohumeral joint, characterised by pain and uniform limitation of all movements, with a tendency to slow spontaneous recovery.

Cause. This is unknown. There is no evidence of a bacterial infection. Injury is an inconstant factor and its significance is doubtful

Pathology. This is not understood : it presents a baffling problem It is believed that there is a loss of resilience of the joint capsule, but the nature of the underlying changes has not been explained. Whatever their nature, the changes are reversible, for in most cases the joint is eventually restored almost to normal.

Clinical features. The patient complains of severe aching pain in the shoulder and upper arm of gradual and spontaneous onset. *On examination* the only finding is a uniform impairment of all gleno-humeral movements—abduction, flexion, extension, rotation—which are often reduced to about a quarter or half of their normal range. In a severe case most of the shoulder movement that remains is contributed by scapular movement, which is unimpaired. *Radiographs* do not show any abnormality.

Diagnosis. Other causes of painful limitation of gleno-humeral movement, especially the various forms of arthritis, must be excluded by careful clinical and radiographic examination. The characteristic feature of "frozen" shoulder is the uniform limitation of all gleno-humeral movements without evidence of inflammatory or destructive changes.

Course. There is a tendency towards spontaneous recovery, usually within six to twelve months. The pain subsides first, leaving gleno-humeral joint stiffness, which thereafter gradually diminishes with active use of the limb. If movements are not practised deliberately some permanent restriction of movement may remain.

Treatment. In the early, acutely painful stage the arm is rested in a sling, which is removed for short periods each day to permit gentle assisted shoulder exercises. Cortisone (or ACTH) is often effective in reducing pain and permitting earlier restoration of movement. When the pain lessens, active exercises are intensified and continued for weeks or months until full movement is regained. If mobilisation is very slow after the pain has abated the shoulder should be manipulated gently under anaesthesia.

TENOSYNOVITIS OF THE LONG BICEPS TENDON

(Biceps tendinitis)

This is an uncommon affection characterised by pain and local tenderness in the region of the bicipital groove of the humerus and the long tendon of the biceps.

Pathology. The condition is generally attributed to frictional irritation of the tendon within its groove.

Clinical features. The complaint is of pain in the front of the shoulder, worse on active use of the arm. Examination reveals local tenderness in the course of the long tendon of the biceps.

The pain can often be exacerbated by moving the shoulder while the tendon is tautened by forced supination of the forearm.

Treatment. Excessive use of the shoulder should be avoided, and in severe cases a sling may be worn for part of the day. A course of short-wave diathermy to the tender area often seems to hasten recovery.

DISORDERS OF THE ACROMIO-CLAVICULAR JOINT

OSTEOARTHRITIS OF THE ACROMIO-CLAVICULAR JOINT

Though not common, acromio-clavicular osteoarthritis is seen much more often than is osteoarthritis of the gleno-humeral joint. Pathologically, there is degeneration and attrition of articular cartilage, and spurs of bone (osteophytes) are formed at the joint margins.

Clinical features. There is pain, localised accurately to the acromio-clavicular joint and aggravated by strenuous use of the limb—especially by overhead work. *On examination* irregular bony thickening of the joint margins due to osteophytes can be felt There is no soft-tissue thickening and no increase of local skin temperature The total range of shoulder movements is not appreciably decreased, but the local acromio-clavicular pain is exacerbated at the extremes of movement. This is most easily demonstrated during abduction of the arm : the arc of movement below 90 degrees is painless, but above 90 degrees pain develops and persists throughout the remainder of the arc to full elevation (compare painful arc syndrome). *Radiographs* show narrowing of the cartilage space and marginal osteophytes.

Treatment. Often no treatment is needed. Conservative treatment is by short-wave diathermy. In severe cases operative treatment is justified. It should take the form of excision of the lateral end of the clavicle or removal of the acromion.

PERSISTENT ACROMIO-CLAVICULAR DISLOCATION OR SUBLUXATION

Persistent upward displacement of the lateral end of the clavicle is a common sequel to traumatic dislocation or subluxation of the acromio-clavicular joint In most cases the displacement

is slight and causes no symptoms. Exceptionally there is pain, worse during full elevation of the arm. *On examination* the lateral end of the clavicle is unduly prominent, and a distinct step can be felt between it and the surface of the acromion.
Treatment. Usually no treatment is required. Rest in a sling for a few days is sufficient to relieve a temporary exacerbation of pain brought on by over-use of the arm. If disabling pain persists, operation is advised. A simple and effective method is to excise the lateral end of the clavicle.

DISORDERS OF THE STERNO-CLAVICULAR JOINT

PYOGENIC ARTHRITIS OF THE STERNO-CLAVICULAR JOINT

Organisms may reach the joint through the blood stream or, rarely, through a penetrating wound. There is acute or subacute inflammation, with exudation of fluid into the joint or, in severe cases, suppuration and abscess formation.
Clinical features. The symptoms are local swelling and pain. The pain is aggravated by active use of the arm. *On examination* there is diffuse swelling over the joint. The overlying skin is abnormally warm and it may be reddened. The total range of shoulder movements is slightly impaired and there is pain at the extremes.
Radiographs show no abnormality in the early stages, but if the lesion advances local osteoporosis and narrowing of the cartilage space are to be expected
Treatment. Whenever possible the causative organism is identified and the appropriate antibiotic drug is administered. The limb is rested in a sling A joint effusion or abscess is aspirated or, in suppurative cases, drained by incision.

TUBERCULOUS ARTHRITIS OF THE STERNO-CLAVICULAR JOINT

Tubercle bacilli reach the joint through the blood stream from a focus elsewhere As in other tuberculous joints, the synovial membrane is thickened and, if the disease advances, articular cartilage and bone are eroded.
Clinical features. The symptoms are local swelling and pain.

The pain can often be exacerbated by moving the shoulder while the tendon is tautened by forced supination of the forearm.

Treatment. Excessive use of the shoulder should be avoided, and in severe cases a sling may be worn for part of the day. A course of short-wave diathermy to the tender area often seems to hasten recovery.

DISORDERS OF THE ACROMIO-CLAVICULAR JOINT

OSTEOARTHRITIS OF THE ACROMIO-CLAVICULAR JOINT

Though not common, acromio-clavicular osteoarthritis is seen much more often than is osteoarthritis of the gleno-humeral joint. Pathologically, there is degeneration and attrition of articular cartilage, and spurs of bone (osteophytes) are formed at the joint margins.

Clinical features. There is pain, localised accurately to the acromio-clavicular joint and aggravated by strenuous use of the limb—especially by overhead work. *On examination* irregular bony thickening of the joint margins due to osteophytes can be felt. There is no soft-tissue thickening and no increase of local skin temperature. The total range of shoulder movements is not appreciably decreased, but the local acromio-clavicular pain is *exacerbated at the extremes of movement. This is most easily* demonstrated during abduction of the arm: the arc of movement below 90 degrees is painless, but above 90 degrees pain develops and persists throughout the remainder of the arc to full elevation (compare painful arc syndrome). *Radiographs* show narrowing of the cartilage space and marginal osteophytes.

Treatment. Often no treatment is needed. Conservative treatment is by short-wave diathermy. In severe cases operative treatment is justified. It should take the form of excision of the lateral end of the clavicle or removal of the acromion.

PERSISTENT ACROMIO-CLAVICULAR DISLOCATION OR SUBLUXATION

Persistent upward displacement of the lateral end of the clavicle is a common sequel to traumatic dislocation or subluxation of the acromio-clavicular joint. In most cases the displacement

DISORDERS OF THE BRACHIAL PLEXUS OR ITS ROOTS

The pain caused by pressure upon the brachial plexus or its roots is commonly attributed erroneously to an affection of the shoulder. Such pain varies in its precise distribution according to the site and nature of the lesion. Usually it radiates from the base of the neck, across the top of the shoulder, and down the front, side, or back of the arm; thence it extends into the forearm, and often into the hand and fingers. Thus in its typical form the pain of a brachial plexus lesion differs from the pain of a shoulder lesion, which does not extend below the elbow.

The commonest affections to cause referred symptoms in the distribution of the brachial plexus are prolapsed cervical intervertebral disc, osteoarthritis of the cervical spine, cervical rib, herpes zoster, and tumours involving the spinal cord or the component nerves of the brachial plexus. These conditions were described in Chapter III.

DISORDERS WITHIN THE THORAX

Angina Pectoris

In a small proportion of cases of angina pectoris the pain is felt predominantly in the shoulder region (usually on the left side). Other features are invariably present to suggest a cardiac origin, and the shoulder shows no clinical abnormality. If the history and findings are elicited with care there is little difficulty in distinguishing cardiac pain from true shoulder pain.

Pleurisy

Basal pleurisy is sometimes a cause of shoulder pain, which is explained by irritation of phrenic nerve endings, with referred pain in the distribution of the cutaneous branches of the same cervical roots (mainly C 4). The shoulder is clinically normal, and the other features of the disease are usually sufficiently clear to indicate its true nature.

DISORDERS WITHIN THE ABDOMEN

Cholecystitis

This is a cause of referred pain in the right shoulder, from irritation of the phrenic nerve endings under the diaphragm. The

The pain is worse after active use of the limb. *On examination* there is a diffuse swelling over the joint. The overlying skin is warmer than normal. An abscess or discharging sinus may be present. Shoulder movement causes pain at the extremes and the total range is restricted. *Radiographs* show osteoporosis adjacent to the sterno-clavicular joint. Later, if the disease advances, there is loss of cartilage space and erosion of bone at the joint surface.

Treatment. The principles of treatment are the same as for other tuberculous joints (p. 40).

PERSISTENT OR RECURRENT DISLOCATION OF THE STERNO-CLAVICULAR JOINT

Forward dislocation of the medial end of the clavicle may be permanent, or it may recur on certain movements of the limb. Often, but not always, there is a history of precipitating injury. The symptoms are slight: there is a prominence in the region of the joint, with mild local pain. Recurrent displacement of the clavicle in and out during movements of the arm may be an annoying disability. *On examination* the medial end of the clavicle, when displaced, is easily felt as a prominent forward projection. In recurrent dislocation the clavicle can be felt to click out of joint when the shoulders are braced back and to go back into position when the shoulders are arched forwards. *Radiographs* reveal the displacement, when present. The joint is difficult to show clearly and special projections are necessary.

Treatment. In many cases treatment is unnecessary. Non-operative treatment is ineffective. Operation is occasionally justified: the displacement is reduced and the clavicle is held in place by constructing a new retaining ligament from the tendon of the subclavius muscle or from a strip of fascia.

EXTRINSIC DISORDERS SIMULATING SHOULDER DISEASE

Pain in the shoulder or arm often has no local cause, but is referred from an extrinsic lesion. Such a possibility must always be considered in differential diagnosis

CHAPTER SIX

The Upper Arm and Elbow

APART from injury, disorders of the upper arm and elbow region are generally straightforward and present few special problems. They conform to the general descriptions of bone and joint diseases that were given in Chapter II. Thus the humerus is subject to the ordinary infections of bone, and occasionally to bone tumours—especially metastases. The elbow is liable to every type of arthritis, though none is particularly common. After the knee, it is the joint most often affected by osteochondritis dissecans and loose body formation. The ulnar nerve lies in a vulnerable position at the back of the medial epicondyle, and the possibility of impairment of nerve function complicating disease or injury of the joint should always be remembered.

SPECIAL POINTS IN THE INVESTIGATION OF UPPER ARM AND ELBOW SYMPTOMS

History

The interrogation follows the usual lines suggested in Chapter I. It is important to ascertain the exact site and distribution of the pain, and its nature. Pain arising locally in the humerus is easily confused with pain arising in the shoulder, which characteristically radiates to a point about half-way down the outer aspect of the arm. Elbow pain is localised fairly precisely to the joint, though a diffuse aching pain is often felt also in the forearm. When the ulnar nerve is interfered with behind the elbow the symptoms are predominantly in the hand.

In the elbow, a history of previous injury, perhaps long ago in childhood, is often significant. Injuries in this region are notoriously liable to have late effects in the form of impaired movement, deformity, arthritis, loose body formation, or interference with the ulnar nerve.

Exposure

The whole length of the upper limb must be uncovered. The opposite limb must be similarly exposed for comparison,

associated abdominal symptoms and signs, and the lack of clinical abnormality in the shoulder, should prevent diagnostic errors.

Subphrenic Abscess

This also is an occasional cause of referred shoulder pain. Constitutional symptoms and pyrexia, with normal clinical findings in the shoulder, exonerate the shoulder from blame.

ends of the radius and ulna (allowing rotation of the forearm). It should be remembered that free rotation of the forearm is dependent not only upon an intact superior radio-ulnar joint; it demands also free mobility between radius and ulna throughout their length, and at the inferior radio-ulnar joint. *Flexion-extension:* The normal range is from 30 to 180 degrees. *Supination-pronation:* Rotation movements must be tested with the elbow flexed to a right angle, to eliminate rotation at the shoulder (Fig. 150). The normal range is 90 degrees of supination (palm up) and 90 degrees of pronation (palm down). If the range of rotation is restricted possible causes must be sought in the forearm and wrist as well as in the elbow.

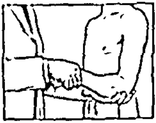

FIG. 150

Examining forearm rotation. The elbows are flexed 90 degrees to eliminate rotation at the shoulder.

The Ulnar Nerve

Because of the vulnerability of the ulnar nerve in its course behind the elbow, tests of ulnar nerve function should be carried out as part of the routine examination of the elbow. Examine for sensibility in the little finger and medial half of the ring finger, and test the ulnar-innervated small muscles of the hand for wasting or weakness. Note whether the skin in the territory of the ulnar nerve sweats equally with the rest of the hand.

Radiographic Examination

Radiographs of the humerus must always include antero-posterior and lateral projections, and they should take in both the shoulder joint and the elbow.

Routine radiographs of the elbow comprise an antero-posterior projection with the elbow straight and a lateral projection with the joint semi-flexed In special circumstances additional oblique or tangential projections may be helpful A radiograph of the forearm bones and of the inferior radio-ulnar joint is also required when forearm rotation is impaired

Extrinsic Sources of Pain in the Upper Arm

Pain in the upper arm is commonly referred from a lesion elsewhere —particularly from the shoulder, and from the neck when the brachial plexus or its roots are involved. Shoulder pain usually radiates from the tip of the acromion process to about the middle of the outer aspect of the arm, but it does not extend below the elbow. In contrast, nerve pain from interference with the brachial plexus often extends *throughout* the length of the arm and forearm into the hand and fingers; and frequently there are accompanying paraesthesiae in the form of tingling, numbness, or "pins and needles."

Steps in Routine Examination.

A suggested plan for the routine clinical examination of the upper arm and elbow is summarised in Table VI.

TABLE VI

ROUTINE CLINICAL EXAMINATION IN SUSPECTED DISORDERS OF THE UPPER ARM AND ELBOW

1. LOCAL EXAMINATION OF THE ARM AND ELBOW

Inspection	**Power**
Bone contours and alignment	Flexors
Soft-tissue contours	Extensors
Colour and texture of skin	Supinators
Scars or sinuses	Pronators
Palpation	**Stability**
Skin temperature	Lateral ligament
Bone contours	Medial ligament
Soft-tissue contours	
Local tenderness	**The ulnar nerve**
Movements (active and passive)	Sensory function
Humero-ulnar joint	Motor function
Flexion	Sweating
Extension	
Radio-ulnar joint .	
Supination	
Pronation	
? Pain on movement	
? Crepitation on movement	

2 EXAMINATION OF POTENTIAL EXTRINSIC SOURCES OF ARM PAIN

This is important if a satisfactory explanation for the symptoms is not found on local examination. The investigation should include · 1) the neck, with the brachial plexus , and 2) the shoulder.

3 GENERAL EXAMINATION

General survey of other parts of the body The local symptoms may be only one manifestation of a widespread disease.

Movements at the Elbow

The elbow joint has two distinct components . the hinge joint between the humerus above and the ulna and radius below (allowing flexion-extension movement) , and the pivot joint between the upper

CLASSIFICATION OF DISORDERS OF THE ARM AND ELBOW

DISORDERS OF THE UPPER ARM

INFECTIONS

Acute osteomyelitis
Chronic osteomyelitis

TUMOURS

Benign tumours of bone
Malignant tumours of bone

DISORDERS OF THE ELBOW

DEFORMITIES

Cubitus valgus
Cubitus varus

ARTHRITIS

Pyogenic arthritis
Rheumatoid arthritis
Tuberculous arthritis
Osteoarthritis
Haemophilic arthritis
Charcot's osteoarthropathy

MECHANICAL DERANGEMENTS

Osteochondritis dissecans
Loose bodies in the elbow

EXTRA-ARTICULAR DISORDERS

"Tennis" elbow
Olecranon bursitis
Friction neuritis of the ulnar nerve

infrequently. Metastatic tumours, by comparison, are common, especially in the proximal part of the humerus.

OSTEOGENIC SARCOMA

The upper metaphysis of the humerus is the favourite site in the upper limb for this highly malignant tumour. It occurs only exceptionally at the lower metaphysis. The tumour affects children or young adults and has the usual characteristics of such tumours, destroying the metaphysial region and bursting out through the cortex to invade the adjacent soft tissues. Metastasising early by the blood stream, its cells quickly take root in the lungs. Even despite early amputation the outcome is nearly always fatal from metastases.

EWING'S TUMOUR

This occurs occasionally in the shaft of the humerus but it is very uncommon. In its behaviour it conforms to the general description given in Chapter II.

MULTIPLE MYELOMA

The tumour foci of multiple myeloma develop readily in the proximal half of the humerus, which contains abundant vascular marrow.

METASTATIC TUMOURS

Carcinomatous deposits from tumours of the lung, breast, prostate, kidney, and thyroid are common in the humerus They usually occur near the upper end of the shaft, where there is much vascular marrow. Such metastases are a common cause of pathological fracture in the upper limb A typical example is shown in Figure 152.

FIG 152
Metastatic tumour in the humerus, with pathological fracture

positive in the incipient stage. There is a marked polymorphonuclear leucocytosis.

Treatment. This is the same as for acute osteomyelitis elsewhere. The main principles are rest and chemotherapy, with immediate drainage if an abscess should form.

CHRONIC OSTEOMYELITIS

(General description of chronic osteomyelitis, p. 59.)

As in other bones, chronic pyogenic osteomyelitis of the humerus is nearly always a sequel to acute osteomyelitis that has been neglected or has responded poorly to treatment. The bone is thickened, often throughout its whole length, and there is frequently a persistent or intermittent purulent discharge from a sinus. *Radiographs* show irregular thickening with patchy areas of sclerosis and cavitation, and sometimes a sequestrum.

TUMOURS OF BONE

BENIGN TUMOURS

(General description of benign bone tumours, p. 66.)

OSTEOCLASTOMA

This is the only benign tumour of much practical importance in this region Though it is not common at any site, it occurs relatively frequently at the upper end of the humerus, where it often extends close up to the articular surface The tumour occurs chiefly in young adults, and its general characteristics are like those of osteoclastoma elsewhere (p. 69).

Treatment. The risk of recurrence after simple curettage or radiotherapy has to be balanced against the likely disability after radical excision of the whole of the upper end of the humerus. Each case must be considered on its merits and every factor taken into consideration

MALIGNANT TUMOURS

(General description of malignant bone tumours, p. 70.)

Primary malignant tumours of bone are much less common in the upper limb than in the lower, and examples are seen only

CUBITUS VARUS

Cubitus varus is the opposite deformity to cubitus valgus. The "carrying angle," or normal angle of valgus at the fully extended elbow, is decreased or reversed.

Cause. The causes are similar to those of cubitus valgus: 1) previous fracture with mal-union; and 2) interference with epiphysial growth on the medial side.

Clinical features. There are usually no symptoms other than the visible deformity. Osteoarthritis is an occasional sequel in long-established cases.

Treatment. Minor degrees of deformity can safely be left uncorrected. If the angulation is marked it may be corrected by osteotomy through the lower end of the humerus.

PYOGENIC ARTHRITIS OF THE ELBOW

(General description of pyogenic arthritis, p. 31.)

Pyogenic arthritis is usually an acute infection with suppuration, though it may occur in subacute or even chronic form.

Pathology. The organisms reach the joint in three ways: 1) through the blood stream (haematogenous infection); 2) through a penetrating wound; or 3) from an adjacent focus of osteomyelitis in the humerus, radius, or ulna. There is an acute or subacute inflammatory reaction, with exudation of fluid into the joint; the fluid is turbid or frankly purulent according to the severity of the infection. The outcome varies from complete healing, with restoration of normal function, to total destruction of the joint with fibrous or bony ankylosis.

Clinical features. The onset is acute or subacute, with pain and swelling of the elbow. There is constitutional illness, with pyrexia *On examination* the elbow is swollen (partly fluid, partly synovial thickening). The overlying skin is warmer than normal and it may be reddened. All movements are limited by pain and muscle spasm A primary focus of infection is often found elsewhere in the body. *Radiographs* show no change in the early stages Later, if the infection persists, there may be diffuse osteoporosis and loss of cartilage space *Investigations:* The erythrocyte sedimentation rate is raised. There is a polymorphonuclear leucocytosis. Bacteriological examination of aspirated pus or fluid usually reveals the identity of the causative organism.

DISORDERS OF THE ELBOW

CUBITUS VALGUS

The normal elbow, when fully extended, is in a position of slight valgus—usually 10 degrees in men and 15 degrees in women This is known as the "carrying angle." If the angle is increased, so that the forearm is abducted excessively in relation to the upper arm, the deformity is known as cubitus valgus (Fig. 153).

Cause. Cubitus valgus is usually a consequence of previous disease or injury in the elbow region. The most frequent causes are: 1) previous fracture of the lower end of the humerus, with mal-union; and 2) interference with epiphysial growth on the lateral side, from injury or infection.

Clinical features. Apart from the visible deformity there are no symptoms unless secondary effects develop.

Secondary effects. The most important sequel of cubitus valgus is interference with the function of the ulnar nerve. When valgus deformity is marked, the nerve is angled sharply round the prominent medial part of the joint, and repeated friction may lead to fibrosis of the nerve trunk. Symptoms develop insidiously over a long period: there are tingling and blunting of sensation in the ulnar distribution in the hand, with weakness and wasting of the ulnar-innervated small hand muscles (p. 236)

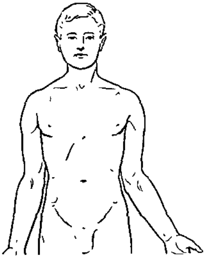

FIG. 153

Cubitus valgus. The deformity predisposes to friction neuritis of the ulnar nerve

Long-established cubitus valgus may lead to osteoarthritis of the elbow, especially in those who do heavy work.

Treatment. Slight uncomplicated *deformity is best left alone.* If angulation is severe, correction by osteotomy near the lower end of the humerus is justified If the function of the ulnar nerve is impaired the nerve should be transposed from its post-humeral groove to a new bed at the front of the elbow.

Operative treatment: If extensive destruction of the articular cartilage leads to persistent disabling pain operation is worth considering. Useful function, with relief of pain, can often be restored by arthroplasty of the excision type (p. 20).

TUBERCULOUS ARTHRITIS OF THE ELBOW

(General description of tuberculous arthritis, p. 37.)

Tuberculous arthritis is uncommon in the elbow when compared with its incidence in the large weight-bearing joints such as the hip and knee.

Pathology. The pathological features conform to the general pattern of tuberculous arthritis. The synovial membrane is much thickened by inflammatory changes of tuberculous type. Unless the disease is arrested, the articular cartilage is soon destroyed and the underlying bone eroded. A tuberculous abscess commonly forms, and it may discharge at the skin surface to form a chronic sinus.

Clinical features. Children and young adults are most commonly affected. The symptoms are pain in the elbow, swelling, and impaired movement. *On examination* the joint is diffusely swollen by synovial thickening. The overlying skin is warmer than normal. All movements are restricted, and attempts to force movements aggravate the pain An abscess or discharging sinus may be evident Often a tuberculous lesion is present elsewhere in the body *Radiographic examination* . The earliest change is diffuse osteoporosis throughout the area of the elbow. Later, if the disease progresses, the cartilage space is narrowed and the underlying bone eroded *Investigations :* The erythrocyte sedimentation rate is increased The Mantoux test is positive. Biopsy of the synovial membrane reveals the typical histological features of tuberculosis

Diagnosis. Tuberculous arthritis of the elbow has to be distinguished from other forms of subacute or chronic arthritis. When doubt exists, biopsy of the synovial membrane should be undertaken to establish the diagnosis as early as possible.

Course. Under favourable conditions there is a natural tendency to slow healing by fibrosis. The usual outcome is a fibrous ankylosis with a few degrees of movement , but with modern

Treatment. Systemic chemotherapy must be begun at the earliest moment. Whenever possible the causative organism must be identified and its sensitivity to antibiotics determined, so that the most effective drug can be given. Meanwhile the joint is rested in a split plaster, and the joint fluid is aspirated repeatedly or, when necessary, drained through an incision. After each aspiration the appropriate antibiotic drug is injected into the joint Rest is continued until the infection has been overcome ; thereafter active movements are encouraged.

RHEUMATOID ARTHRITIS OF THE ELBOW

(General description of rheumatoid arthritis, p. 34.)

One or both elbows are commonly affected in rheumatoid arthritis, usually in conjunction with several other joints.

Pathology. The pathological changes are like those of rheumatoid arthritis elsewhere. Beginning as a chronic inflammatory thickening of the synovial membrane, it tends later to involve the articular cartilage, which may eventually be almost totally destroyed.

Clinical features. As in other joints, the main symptoms are pain, swelling, and stiffness. The pain is worse when activity is first resumed after resting. *On examination* the affected elbow is swollen from synovial thickening. The overlying skin is warmer than normal The range of flexion, extension, and rotation is limited and causes pain, especially at the extremes. The controlling muscles are often wasted. The changes in other affected joints are similar *Radiographic examination :* At first there are no changes. Later, there is diffuse osteoporosis in the area of the joint. In long-established cases the cartilage space is lost and there may be some erosion of the bone ends (Fig. 20). *Investigations :* The erythrocyte sedimentation rate is increased during the active phase.

Course. There is a tendency for the disease to become inactive after a variable course lasting for months or years. The joint is seldom restored to normal , usually there is moderate permanent restriction of movement, with a tendency to pain brought on by heavy use of the limb.

Treatment. Primary treatment is along the lines suggested for rheumatoid arthritis in general

On examination there is palpable thickening at the margins of the joint from osteophytes. Flexion and extension are impaired but rotation is often full. There is coarse crepitation on movement. *Radiographs* show narrowing of the cartilage space and pointed osteophytes at the joint margins. There is a tendency to sclerosis of bone at the joint surfaces (Fig. 154). Loose bodies (formed from detached osteophytes) may be present

FIG. 154

Osteoarthritis of the elbow. Note the narrowed cartilage space and pointed osteophytes at the joint margins. In this case osteoarthritis was secondary to osteochondritis dissecans

Treatment. In many cases no treatment is required once the nature of the trouble has been explained to the patient. When treatment is called for conservative measures are tried first. Physiotherapy in the form of short-wave diathermy is usually adequate, especially if heavy use of the elbow can be reduced. Only exceptionally is operation justified: the methods available are arthroplasty (by excision of the joint surfaces) and arthrodesis

If a loose body has caused symptoms of locking, it should be removed by operation

HAEMOPHILIC ARTHRITIS OF THE ELBOW

(General description of haemophilic arthritis, p. 46)

Haemophilic arthritis affects the elbow more often than any other joint except the knee. As in other joints, the main feature is intra-articular haemorrhage, with consequent irritation and, later, degeneration of the joint.

Clinical features. The condition is confined to males. The patient may or may not be a known sufferer from haemophilia. There is often a history of previous haemorrhages. The elbow is painful and swollen Movements are restricted. *Radiographs* show no abnormality in the early stages *Investigations:* In most

treatment the joint can sometimes be preserved virtually intact, especially in children.

Treatment. The patient should be admitted to a country orthopaedic hospital. *Constitutional treatment :* This is by rest and systemic chemotherapy (streptomycin, para-amino-salicylic acid, and isonicotinic acid hydrazide for six months if well tolerated). *Local treatment :* Initial treatment is by immobilisation in plaster for six months while the body builds up a resistance against the disease. Thereafter treatment depends upon the damage sustained by the joint. If after six months there is no evidence of destruction of articular cartilage or bone there is a reasonable chance that the disease has been arrested, and active elbow movements may be begun. But if cartilage is destroyed or *bone eroded the joint can never be restored to normal.* In these circumstances rest in plaster should be continued for as long as is necessary to allow the disease to become inactive. The resulting fibrous ankylosis often allows reasonably satisfactory function without pain. But in a proportion of cases the disease is reactivated, pain returns, and operation is required. *Operation :* In the elbow, unlike most other joints, simple excision of the bone ends is capable of restoring satisfactory function. The false joint is rather flail and unstable, but nevertheless has useful movement. This operation is a form of arthroplasty. The alternative is arthrodesis. The choice depends largely upon the type of work to be demanded of the limb

OSTEOARTHRITIS OF THE ELBOW

(General description of osteoarthritis, p. 41.)

Osteoarthritis of the elbow seldom occurs without predisposing injury or disease of the joint surfaces.

Cause. It is caused by wear and tear. But in nearly every case a predisposing factor has been present for several years. This is usually a damaged articular surface from previous fracture involving the joint or from osteochondritis dissecans.

Clinical features. There is slowly increasing pain in the elbow, worse on heavy use of the limb The patient may also notice that movement is impaired. In some cases there are attacks of sudden locking, suggesting the presence of a loose body in the joint. There is often a history of previous injury involving the elbow.

Pathology. The protective mechanism which prevents the normal joint from being damaged by everyday stresses fails because the ligaments are insensitive to pain. A vicious circle is established: repeated unrecognised injuries impair the stability of the joint and thereby render it more liable to further injury. The ultimate result is gross disorganisation. Basically, the changes consist of degeneration and attrition of the joint surfaces, sometimes with coincident massive hypertrophy of bone at the joint margins. Pathological dislocation may occur (Fig. 155).

Clinical features. The main symptoms are swelling and a feeling of weakness due to instability. In the early stages pain may not be entirely absent, but in general lack of pain is a striking feature. *On examination* there is marked thickening and irregularity of the bone ends. The joint is abnormally lax, and lateral mobility is often pronounced. Clinical evidence of the underlying condition (usually syringomyelia) will be found. *Radiographs* show a disorganised joint, often with much destruction of bone (Fig. 155). *Investigations* should be directed towards establishing the nature of the underlying condition.

Treatment. This is mainly that of the underlying condition. If the elbow is severely disorganised it should be protected by a right-angled splint of plastic or leather. Exceptionally, arthrodesis is justified

OSTEOCHONDRITIS DISSECANS OF THE ELBOW

(General description of osteochondritis dissecans, p. 52.)

After the knee, the elbow is the most frequent site of osteochondritis dissecans. The disorder is characterised by necrosis of part of the articular cartilage and of the underlying bone, with eventual separation of the fragment to form an intra-articular loose body.

Cause. The precise cause is unknown. Impairment of blood supply to the affected segment of bone and cartilage by thrombosis of an end-artery has been suggested. Injury probably plays a part. There is thought to be an inborn constitutional susceptibility to the disease, for it may occur in several joints of the same patient, or in several members of a family.

types of haemophilia the clotting time of the blood is increased. Aspiration confirms the presence of blood in the joint.

Diagnosis. The clue to the diagnosis is a history of previous bleeding or of a haemophilic tendency in the family. Haemarthrosis without major injury is suggestive and should arouse suspicion of haemophilia. A prolonged blood-clotting time is an important confirmatory sign.

Treatment. General treatment is the same as that for other manifestations of haemophilia, including blood transfusions when necessary. Local treatment is by firm bandaging and rest in a plaster splint for four weeks, or longer if the tendency to haemarthrosis persists.

CHARCOT'S OSTEOARTHROPATHY OF THE ELBOW

(General description of Charcot's osteoarthropathy, p. 47.)

In Charcot's disease of the elbow the joint becomes disorganised in consequence of a loss of sensibility to pain.

FIG. 155

Charcot's osteoarthropathy of the elbow. There is marked absorption of bone, with pathological dislocation. The underlying cause was syringomyelia.

Cause. The commonest underlying cause of the disease in the elbow is syringomyelia.

LOOSE BODIES IN THE ELBOW

Causes. There are four important causes of loose bodies in the elbow: 1) osteochondritis dissecans (1 to 3 bodies); 2) osteoarthritis (1 to 3 bodies); 3) fracture with separation of a fragment (1 to 3 bodies); and 4) osteochondromatosis (50 to 500 bodies).

FIG. 157
Multiple loose bodies in the elbow. A case of osteochondromatosis.

Pathology and clinical features. *Osteochondritis dissecans* was described on page 231 and *osteoarthritis* on page 228. *Loose body after fracture:* The fragment is usually detached from the capitulum. Sometimes the medial epicondyle is detached and "sucked" into the joint, retaining its attachments to the flexor muscles. *Osteochondromatosis:* This is a rare disease of synovial membrane in which numerous synovial villi become pedunculated and transformed into cartilage; eventually they are detached to form a large number of loose bodies, many of which become ossified. **Clinical features.** Many so-called loose bodies are "silent"—that is, they cause no symptoms. Often in such cases the fragment is not in fact loose, but has soft-tissue attachments which prevent its moving about the joint.

The characteristic symptom of a freely movable loose body is sudden locking of the elbow during movement, with intense pain. The joint is usually "unlocked" after an interval, either spontaneously or by the patient's manoeuvres. Several hours later

Pathology. The affected segment of the articular surface varies in size; commonly its surface area is about half an inch in diameter and its depth about a quarter of an inch. The part of the elbow most often affected is the capitulum. Within the area of the lesion bone and cartilage are avascular, and a line of demarcation forms between the avascular segment and the surrounding normal bone and cartilage. After an interval of months the avascular segment separates as a loose body (sometimes two or three), leaving a shallow cavity in the articular surface which is ultimately filled with fibrous tissue The damage to the joint surface predisposes to the later development of osteoarthritis.

Clinical features. In the early stages (before the fragment has separated) the symptoms are those of mild mechanical irritation of the joint—namely, aching after use and intermittent swelling. *On examination* at this stage there is often a little swelling from effusion of clear fluid into the joint, and there is slight limitation of flexion or extension. In the stage of loose body the main features are recurrent painful locking of the elbow followed by effusion of fluid. *Radiographic examination:* In the early stages there is an area of irregularity on the affected articular surface (usually the capitulum). Later a shallow cavity, whose margins are demarcated clearly from the bone within it, is seen (Fig. 156). Eventually the bony fragment separates from the cavity and lies free within the joint, usually in the lateral compartment.

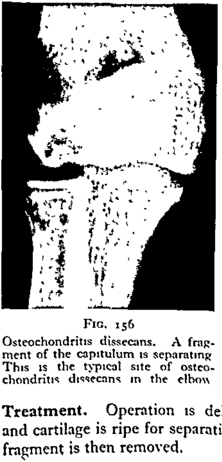

FIG. 156

Osteochondritis dissecans. A fragment of the capitulum is separating This is the typical site of osteochondritis dissecans in the elbow

Treatment. Operation is delayed until the fragment of bone and cartilage is ripe for separation or has actually separated. The fragment is then removed.

Conservative treatment : The following methods are worthy of trial: 1) Physiotherapy, in the form of short-wave diathermy, deep massage to the tender area, and faradic stimulation of the extensor muscles 2) Injection of local anaesthetic into the point of greatest tenderness, with manipulative stretching of the extensor

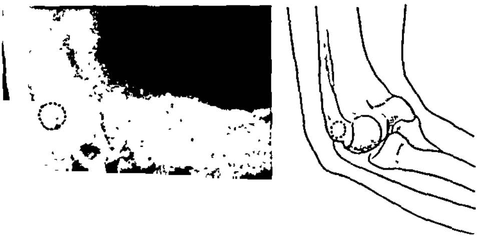

FIG. 158

In " tennis " elbow the point of greatest tenderness is at the front of the lateral epicondyle.

muscles during the period of analgesia (the object of this is to complete the supposed partial rupture of the extensor fibres, in the hope that when they heal the pain will disappear) 3) Injection of hydrocortone into the tender zone *This is claimed to be* capable of producing rapid relief but its efficacy is not yet proved 4) Rest in plaster for six weeks.

Operative treatment : This should be advised only in cases of severe disability not responding to conservative treatment The extensor origin is stripped from its attachment to the lateral epicondyle and allowed to fall back into place After healing the pain is usually found to have disappeared.

OLECRANON BURSITIS

The bursa behind the olecranon is liable to traumatic bursitis, septic bursitis, and gout

In *traumatic bursitis* (" student's elbow ") the bursa is distended

the joint swells. The symptoms subside within a few days, but repeated attacks are to be expected. *Examination* in the stage of swelling shows the joint to be distended with fluid—a clear, pale, straw-coloured effusion. Between attacks a loose body can sometimes be felt. There is often a history or clinical evidence to suggest the nature of the underlying pathology. *Radiographs* show the loose body or bodies (Fig. 157) and usually indicate the nature of the primary condition

Treatment. Symptomless loose bodies can usually be safely left alone ; but if a loose body causes locking it should be removed by operation.

"TENNIS" ELBOW

(Epicondylitis)

"Tennis" elbow is a common and well defined clinical entity. It is an extra-articular affection characterised by pain and tenderness at the site of origin of the extensor muscles of the forearm.

Cause. It is believed to be caused by strain of the forearm extensor muscles at the point of their origin from the bone. Although it sometimes follows tennis, other activities are more frequently responsible.

Pathology. No pathology has been demonstrated. Hypothetically, it is assumed that there is incomplete rupture of aponeurotic fibres at the muscle origin, which is a region plentifully supplied by nerve endings. The elbow joint itself is unaffected.

Clinical features. There is pain at the lateral aspect of the elbow, often radiating down the back of the forearm. *On examination* there is tenderness precisely localised to the front of the lateral epicondyle of the humerus (Fig. 158) Pain is aggravated by putting the extensor muscles on the stretch—for example, by flexing the wrist and fingers with the forearm pronated. Movements of the elbow are full. *Radiographs* show no alteration from the normal.

Course. If left alone the symptoms eventually subside spontaneously, but they may persist for as long as two years.

Treatment. In mild cases the patient is often willing to await spontaneous recovery once the harmless nature of the affection has been explained Treatment is unpredictable in its results and no method can be relied upon in every case.

Conservative treatment : The following methods are worthy of trial: 1) Physiotherapy, in the form of short-wave diathermy, deep massage to the tender area, and faradic stimulation of the extensor muscles. 2) Injection of local anaesthetic into the point of greatest tenderness, with manipulative stretching of the extensor

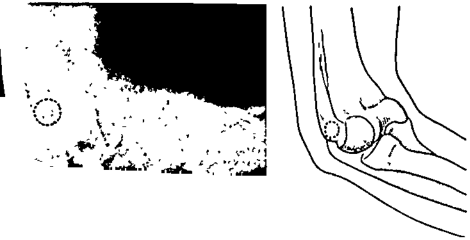

FIG. 158

In " tennis " elbow the point of greatest tenderness is at the front of the lateral epicondyle.

muscles during the period of analgesia (the object of this is to complete the supposed partial rupture of the extensor fibres, in the hope that when they heal the pain will disappear). 3) Injection of hydrocortone into the tender zone This is claimed to be capable of producing rapid relief but its efficacy is not yet proved. 4) Rest in plaster for six weeks.

Operative treatment · This should be advised only in cases of severe disability not responding to conservative treatment. The extensor origin is stripped from its attachment to the lateral epicondyle and allowed to fall back into place After healing the pain is usually found to have disappeared.

OLECRANON BURSITIS

The bursa behind the olecranon is liable to traumatic bursitis, septic bursitis, and gout.

In *traumatic bursitis* (" student's elbow ") the bursa is distended

with clear fluid (Fig. 159). Treatment is by aspiration at first, but if the swelling recurs the bursa should be excised.

Septic bursitis is treated by incision to secure adequate drainage.

FIG. 159
Olecranon bursitis.

In *gouty bursitis* there is acute or subacute inflammation, and whitish deposits of sodium biurate (tophi) may be visible through the walls of the bursa

FRICTION NEURITIS OF THE ULNAR NERVE

The ulnar nerve is vulnerable where it lies in the groove behind the medial epicondyle of the humerus. Its function may be interfered with either by constriction or by recurrent friction while in tension Constriction is usually secondary to osteo-arthritis, with encroachment of osteophytes upon the ulnar groove. Friction under tension occurs when the "carrying angle" of the elbow is increased (cubitus valgus, p. 224) In both cases the nerve undergoes fibrosis, and unless the mechanical fault is relieved the changes become irreversible.

Clinical features. The patient complains of numbness or tingling in the ulnar distribution and often of clumsiness in performing fine finger movements. *On examination* the following signs are present in the fully developed condition · *Sensory*—There is blunting or loss of sensation along the ulnar border of the hand and in the little finger and medial half of the ring finger. *Motor*—There are wasting and weakness of the ulnar-innervated small

hand muscles. *Sweating*—The skin in the ulnar territory is drier than normal, for sweating is impaired.

Treatment. Whenever the ulnar nerve is interfered with by a lesion at the elbow operation should be undertaken to transpose the nerve to a new bed in front of the joint, where it will be free from pressure or friction.

CHAPTER SEVEN

The Forearm, Wrist, and Hand

SO much in everyday life depends upon the efficient working of the hand, and so great is the practical and economic consequence of its disablement, that the care of the diseased or injured hand has become one of the most vital branches of orthopaedic surgery. It is also one of the most fascinating.

Hand surgery is an art and a science in itself. Indeed it is fast developing as a distinct speciality, demanding a knowledge and experience not only of orthopaedics but also of plastic surgery, vascular surgery, and neurology. In America some surgeons are already devoting their whole professional career to work in this field, and there are signs of a similar trend in Great Britain.

In the treatment of hand disorders the primary emphasis should always be on restoration of function. Keen judgment is often called for in deciding between the claims of rest and movement. It should be remembered that the hand tolerates immobilisation badly. Whereas the wrist may be immobilised for many weeks or even months with impunity, to immobilise injured or diseased fingers for a long time is to court disaster in the form of permanent joint stiffness Although rest may be essential in the early days after a hand injury or in the acute stage of an infection, active finger exercises must be insisted upon as soon as that stage is passed It is wise to accept it as a general rule that fingers should never be immobilised for longer than two, or at most three, weeks.

SPECIAL POINTS IN THE INVESTIGATION OF FOREARM, WRIST, AND HAND COMPLAINTS

History

It should be remembered that symptoms in the hand are often caused by disorders of the neck (with involvement of the brachial plexus) and sometimes by disorders at the elbow. Enquiry should

TABLE VII

ROUTINE CLINICAL EXAMINATION IN SUSPECTED DISORDERS OF THE FOREARM, WRIST, AND HAND

1. LOCAL EXAMINATION OF THE FOREARM, WRIST, AND HAND

Inspection

Bone contours
Soft-tissue contours
Colour and texture of skin
Scars or sinuses

Palpation

Skin temperature
Bone contours
Soft-tissue contours
Local tenderness

Movements (active, passive)

At the wrist:

Radio-carpal joint — Flexion-extension; adduction-abduction
Inferior radio-ulnar joint—Supination and pronation

At the hand:

Carpo-metacarpal joint of thumb—Flexion-extension; adduction-abduction, opposition
Metacarpo-phalangeal joints—Flexion-extension; adduction-abduction
Interphalangeal joints—Flexion-extension

Power

Power of each muscle group in control of 1) wrist movement, 2) thumb and finger movement, and 3) gripping

Stability

Tests for abnormal mobility

Nerve function

Tests of sensory function, motor function, and sweating in distribution of median, ulnar and radial nerves

Circulation

Arterial pulses, warmth and colour, capillary return, cutaneous sensibility

2. EXAMINATION OF POSSIBLE EXTRINSIC SOURCES OF FOREARM AND HAND SYMPTOMS

This is important if a satisfactory explanation for the symptoms is not [illegible]

3. GENERAL EXAMINATION

General survey of other parts of the body The local symptoms may be only one manifestation of a more widespread disease.

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Stability

Tests for abnormal mobility

Nerve function

Tests of sensory function, motor function, and sweating in distribution of median, ulnar and radial nerves

Circulation

Arterial pulses, warmth and colour, capillary return, cutaneous sensibility

2. Examination of possible extrinsic sources of forearm and hand symptoms

This is important if a satisfactory explanation for the symptoms is not found on local examination. The investigation should include: 1) the neck and thoracic inlet, with special reference to the brachial plexus; 2) the upper arm, and 3) the elbow

3. General examination

General survey of other parts of the body. The local symptoms may be only one manifestation of a more widespread disease.

always be made into any previous injury or other trouble with the neck or with the upper extremity as a whole.

Exposure

For the local examination the whole forearm should be uncovered to well above the elbow. The sound limb should be exposed likewise for comparison.

Steps in Clinical Examination

A suggested routine of clinical examination is summarised in Table VII.

Movements at the Wrist

Like the elbow, the wrist comprises two distinct components: 1) the radio-carpal joint (including the intercarpal joints), allowing flexion, extension, adduction, and abduction; and 2) the inferior radio-ulnar joint, allowing supination and pronation. The movements at each component must be examined independently.

The radio-carpal joint. The normal range of flexion is 80 degrees and of extension 90 degrees. The range of adduction, or ulnar deviation, is about 35 degrees, and of abduction, or radial deviation, about 25 degrees. It is impracticable to measure the movements of the intercarpal joints individually, and it is simplest to regard them as integral parts of the radio-carpal joint.

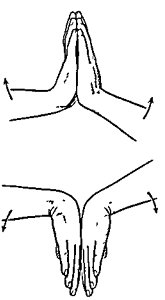

FIG. 160

A simple method of comparing the range of extension and flexion at the two wrists Note the obvious impairment at the left wrist

A rapid and reasonably accurate method of comparing the range of flexion-extension movement on the two sides is as follows: *To judge the range of extension*. The patient places the palms and fingers of the two hands in contact in the vertical plane and lifts the elbows as far as he can while keeping the "heels" of the hands together (Fig. 160). The angle between hand and forearm is easily compared on the two sides. *To judge the range of flexion* · The manoeuvre is reversed. The patient places the backs of the hands together with the fingers directed vertically downwards, and lowers the elbows as far as he can (Fig 160). The angle between hand and forearm is compared on the two sides.

The inferior radio-ulnar joint. The normal range is 90 degrees of supination and 90 degrees of pronation. To determine the range accurately the patient's elbows must be

flexed to a right angle in order to eliminate rotation at the shoulder (Fig. 150, p. 219).

It must be emphasised that impaired rotation does not necessarily denote an abnormality of the wrist: it may equally well be caused by a disorder of the elbow or of the forearm.

Movements of the Hand

The movements of the hand occur mainly at three groups of joints: 1) the carpo-metacarpal joint of the thumb; 2) the metacarpo-phalangeal joints; and 3) the interphalangeal joints.

The carpo-metacarpal joint of the thumb. This joint allows movement in five directions: flexion, or movement of the thumb metacarpal medially in the plane of the palm; extension, or movement of the thumb

FIG. 161

To show the difference between flexion of the thumb across the palm (left) and true opposition (right). In opposition the thumb metacarpal is rotated so that the thumb nail lies in the plane of the palm.

metacarpal laterally in the plane of the palm; adduction, or movement of the metacarpal towards the palm in a plane at right angles to it; abduction, or movement of the metacarpal away from the palm in a plane at right angles to it; and opposition, or rotation of the metacarpal to bring the thumb nail into a plane parallel with the palm (Fig. 161).

[illegible] and fingers. These [illegible] 90 degrees (the range [illegible] f abduction from and adduction to the midline of the middle finger.

The interphalangeal joints of thumb and fingers. These are true hinge joints, allowing only flexion and extension.

Power

Test the power of each movement in turn. In the hand this examination demands considerable patience, for each muscle group must be tested individually. Thus in the thumb it is necessary to test the abductors, the adductor, the extensors (longus and brevis), the flexors (longus and brevis), and the opponens. In the fingers test the flexors (profundus and sublimis), the extensor digitorum and the extensor indicis, the interossei and the lumbricals. *Grip*: Test the power of grip, which demands the combined action of the flexors and extensors of the wrist and the flexors of the fingers and thumb.

16

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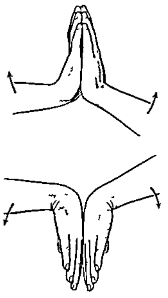

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MISCELLANEOUS

Volkmann's ischaemic contracture
Acute frictional tenosynovitis

ARTICULAR DISORDERS OF THE WRIST AND HAND

DEFORMITIES

Madelung's deformity

ARTHRITIS

Pyogenic arthritis
Rheumatoid arthritis
Tuberculous arthritis
Osteoarthritis

MISCELLANEOUS

Kienböck's disease

EXTRA-ARTICULAR DISORDERS ABOUT THE WRIST AND HAND

INFECTIONS

Acute infections of the fascial spaces
(including digital infective tenosynovitis)
Chronic infective tenosynovitis
(including compound palmar ganglion)

TUMOURS

Tumours of bone
Tumours of soft tissue

NEUROLOGICAL DISORDERS

Compression of the median nerve in the carpal tunnel

MISCELLANEOUS

Ganglion
Dupuytren's contracture
Rupture or severance of tendons
De Quervain's tenovaginitis
Digital tenovaginitis stenosans

Nerve Function

The state of the median, ulnar, and radial nerves is determined tests of sensory function, motor function, and sweating.

Circulation

The state of the circulation is assessed from the condition of t arterial pulses, the warmth and colour of the digits, the capillary retu at the nail beds, and cutaneous sensibility. It should be remember that sensibility to touch in the fingers is a most useful index of t adequacy of the circulation. Nerves require a blood supply to enal them to conduct impulses, and if the circulation is interrupted sensibil is quickly lost.

Extrinsic Sources of Forearm and Hand Symptoms

It is sometimes difficult to determine whether symptoms and sig in the forearm or hand are caused by a local disorder or whether th are referred from a more proximal lesion. This difficulty arises mair in neurological conditions. For instance, the symptoms of compressi of the median nerve in the carpal tunnel may be mimicked closely by prolapsed cervical disc, and the symptoms of constriction of the uln nerve at the elbow may likewise be confused with a low cervical di lesion or a cervical rib. When symptoms in the hand are not satisfactori explained by the local condition a search must be made for a possib cause in the neck, upper arm, or elbow.

Radiographic Examination

Routine radiographs should include antero-posterior and later projections of the forearm, wrist, and hand. For detailed study of t carpal bones additional oblique projections are required.

If it is suspected that the symptoms may be referred from the ne or proximal part of the limb radiographs of the appropriate part shou be obtained.

CLASSIFICATION OF DISORDERS OF THE FOREARM, WRIST, AND HAND

DISORDERS OF THE FOREARM

INFECTIONS OF BONE

Acute osteomyelitis
Chronic osteomyelitis

TUMOURS OF BONE

Benign tumours
Malignant tumours

CHONDROMA

Chondromata of long bones occur chiefly in multiple form, in the condition known as dyschondroplasia, Ollier's disease, or multiple enchondromata (p. 84). Their special significance in the forearm lies in the fact that the tumours may interfere with the normal growth of the affected bone. If growth is retarded in one bone but proceeds normally in its partner a marked curvature of the bones is to be expected and it may cause ugly deformity.

Treatment. Severe deformity from uneven growth of the radius and ulna should be corrected by osteotomy, combined, when necessary, with excision of the lower end of the ulna.

OSTEOCLASTOMA

The lower end of the radius is one of the favourite sites in the upper limb for the development of an osteoclastoma. The lower end of the ulna is also susceptible. The pathology and clinical features are typical of osteoclastoma at other sites. The tumour extends into the former epiphysial region close up to the articular surface.

Treatment. If the lower end of the ulna is the part affected the bone should be excised up to a point well proximal to the tumour. The resulting disability is negligible. If the tumour is in the lower end of the radius treatment is more difficult. Radical excision of the affected part of the bone is the surest safeguard against recurrence of this rather sinister tumour, and it should usually be undertaken despite the inevitable disability that it produces at the wrist. A satisfactory plan of reconstruction after removal of the lower end of the radius is to implant and fuse the lower end of the ulna into the carpus.

MALIGNANT TUMOURS

(General description of malignant tumours of bone, p. 70.)

The radius and ulna are seldom affected by malignant bone tumours, whether primary or metastatic. When osteogenic sarcoma does occur in the forearm the lower end of the radius is the usual site.

DISORDERS OF THE FOREARM

ACUTE OSTEOMYELITIS

(General description of acute osteomyelitis, p. 54.)

Acute osteomyelitis is rather uncommon in the forearm bones. As in other sites, the infection may be blood-borne (haematogenous) or it may be introduced from without, usually in consequence of a compound fracture. The haematogenous type occurs mainly in children. It affects the radius more often than the ulna, and the lower metaphysis rather than the upper. The upper metaphyses of both radius and ulna are partly or wholly within the capsule of the elbow, so an infection of the metaphysis may spread directly to the joint to cause pyogenic arthritis. At the wrist, in contrast, the metaphysis of the radius is wholly outside the capsule, so direct spread of infection to the joint is unlikely. The lower metaphysis of the ulna is partly within the capsule.

The clinical features and treatment are like those of acute osteomyelitis elsewhere.

CHRONIC OSTEOMYELITIS

(General description of chronic osteomyelitis, p. 59.)

As in other bones, chronic osteomyelitis of the radius or ulna follows an acute infection. The pathology and clinical features require no special description.

Treatment. In most cases treatment should follow the usual lines, reliance being placed on rest and chemotherapy for non-suppurative " flares " of infection, and on thorough drainage operations and sequestrectomy for persistent purulent discharge. In obstinate cases the affected length of bone can sometimes be excised without important loss of function : this applies particularly to infection of the lower half of the ulna.

BONE TUMOURS IN THE FOREARM

BENIGN TUMOURS

(General description of benign tumours of bone, p. 66.)

Any type of benign tumour may occur in the forearm bones. Only chondroma and osteoclastoma require further mention here.

in the fingers is obviously impaired. There may or may not be evidence of interruption of nerve conductivity—namely, anaesthesia of the fingers and paralysis of the small hand muscles.

In the established condition, which develops gradually within a few weeks of the injury, there is a striking flexion contracture of the wrist and fingers, from shortening of the fibrotic forearm flexor muscles (Fig. 163). Sensory and motor paralysis of the hand

FIG. 163
Typical appearance of the hand in established Volkmann's ischaemic contracture.

may persist as complicating factors, but they do not form an essential feature of Volkmann's contracture as such.

Diagnosis. In the incipient stage absence of the radial pulse, with marked unwillingness to extend the fingers because of pain, should always arouse suspicion of Volkmann's contracture. If there are also anaesthesia and paralysis of the hand the diagnosis is practically certain. In the established condition the history and clinical features make the diagnosis clear.

Volkmann's ischaemic contracture bears no real resemblance to Dupuytren's contracture (p. 269), for it affects the wrist as well as all the joints of the fingers, and there is no palpable thickening in the palm. Moreover the contracture is demonstrably brought about by shortening of the flexor muscles, for if the wrist is flexed passively to relax the flexor tendons the range of extension at the finger joints is increased. Conversely, if the tendons are relaxed

VOLKMANN'S ISCHAEMIC CONTRACTURE

This is a flexion deformity of the wrist and fingers from fixed contracture of the flexor muscles in the forearm.

Cause. It is caused by ischaemia of the flexor muscles, brought about by injury to, or obstruction of, the brachial artery near the elbow.

Pathology. The effects of sudden occlusion of the brachial artery vary. In a few cases gangrene of the fingers will follow. More often, the collateral circulation is sufficient to keep the hand alive, but not to nourish adequately the flexor muscles of the forearm or the main peripheral nerve trunks. Necrosis of muscle fibres of the forearm flexor group, with subsequent fibrosis and shortening, is the essential feature of Volkmann's contracture. It is often associated with temporary or permanent ischaemic paralysis of the peripheral nerves, especially the median nerve.

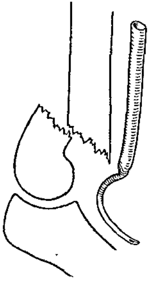

Fig 162

To show how a supracondylar fracture of the humerus may damage the brachial artery, with risk of gangrene or ischaemic contracture Over-tight plaster or dressings may have the same result

The usual primary injury is a supracondylar fracture of the humerus with displacement, the brachial artery being severed or contused by the sharp lower end of the main shaft fragment (Fig 162). Contusion alone is sufficient to interrupt the flow of blood, for the vessel goes into spasm and its lumen may be occluded by thrombosis

In some cases the cause of the arterial obstruction is an over-tight plaster or bandage

Clinical features. The condition is commonest in children. After sustaining a supracondylar fracture of the humerus or some other injury in the elbow region the child complains of pain in the forearm *On examination* in the incipient stage, the fingers are held flexed ; the child is unwilling to straighten them because of the pain caused thereby, but there is no inextensible contracture at this stage. The radial pulse is absent Usually the circulation

impracticable an attempt should be made to restore continuity by an artery graft.

In the established stage restoration to normal is impossible: reconstructive surgery at best can only improve what function remains. The choice of treatment depends upon the circumstances of each case. Measures to counteract the shortening of the flexor muscles include: prolonged stretching by spring devices (applicable only to early cases); shortening of the forearm bones; detachment and distal displacement of the flexor muscle origin; and excision of useless fibrotic muscles. Arthrodesis of the wrist and muscle transfers improve function in selected cases.

ACUTE FRICTIONAL TENOSYNOVITIS

(Peritendinitis; paratendinitis)

This is an easily recognised clinical condition common in young adults whose occupations demand repetitive movements of the wrist and fingers.

Cause. It is attributed to excessive friction between the tendons and the surrounding paratenon, from over-use of the hand. It is entirely distinct from infective tenosynovitis.

Pathology. The tendons most often affected are those of the deep oblique muscles at the back of the forearm, especially the extensor pollicis longus and extensor indicis. There is a mild inflammatory reaction about the tendon and its coverings, with local swelling and oedema.

Clinical features. After unusually active use of the hand over a period of days or weeks pain is felt at the back of the wrist and lower forearm The pain is aggravated by use of the hand. *On examination* there is localised swelling in the line of the affected tendons—usually the extensor pollicis longus and extensor indicis. If the examiner's hand is placed over the swelling while the patient flexes and extends the fingers and thumb a very characteristic fine crepitation is felt (it is caused by the fibrin-covered tendon gliding within the inflamed paratenon). This typical sensation is diagnostic of frictional tenosynovitis.

Treatment. The wrist is immobilised in plaster of Paris for three weeks, the fingers being left free. This affords sufficient rest to allow the inflammation to resolve. Excessive use of the fingers and thumb should be avoided for two months.

by flexing the fingers fully the range of wrist extension is increased (Fig. 164).

Treatment. In the **incipient stage** the problem is that of dealing with a sudden occlusion of the brachial artery. The case must be

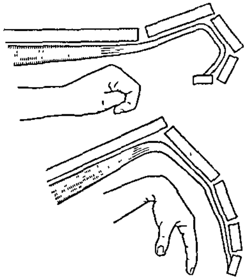

FIG. 164

In Volkmann's contracture the wrist can be partly extended if the fingers are flexed, and the fingers can be partly extended if the wrist is flexed but the shortened fibrotic flexor muscle prevents extension of wrist and fingers together.

handled as an emergency, for the effects of the occlusion become irreversible after a few hours The following action must be taken *First step:* All splints, plaster, and bandages are removed. The limb is rested on a pillow outside the bed and kept cool. Heat cradles or hot bottles are applied over the other three limbs and trunk to promote general vasodilation. If these measures fail to bring about a return of adequate circulation within half an hour, the next step is taken. *Second step:* At operation the brachial artery is explored and the nature of the damage determined. If the occlusion is due to kinking or spasm of the artery an attempt is made to relieve it by freeing the vessel and painting the adventitia with a solution of papaverine. If the vessel is found punctured, or contused and thrombosed, the ideal treatment is to excise the damaged segment and to suture the two ends. If direct suture is

impracticable an attempt should be made to restore continuity by an artery graft.

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ARTICULAR DISORDERS OF THE WRIST AND HAND

MADELUNG'S DEFORMITY

Madelung's deformity is a congenital subluxation or dislocation of the lower end of the ulna. A similar deformity is more often caused by disease or injury, such as a fracture of the lower end of the radius with upward displacement of the lower fragment. The deformity varies in degree from a slight prominence of the lower end of the ulna at the back of the wrist to complete dislocation of the inferior radio-ulnar joint with marked radial deviation of the hand (Fig. 165).

Treatment. If the disability justifies operation, the lower end of the ulna should be excised.

FIG. 165

FIG. 165

Relative shortening of radius with subluxation of inferior radio-ulnar joint and prominent head of ulna (Madelung's deformity)

PYOGENIC ARTHRITIS OF THE WRIST

(General description of pyogenic arthritis, p. 31.)

Pyogenic arthritis of the wrist is uncommon. The infection is usually haematogenous, or it may be introduced through a penetrating wound Spread from a focus of osteomyelitis is rare, partly because osteomyelitis itself is uncommon in the forearm bones, and partly because the lower metaphysis of the radius is entirely outside the capsule of the joint (the lower metaphysis of the ulna is partly intracapsular).

Clinical features. The onset is acute or subacute, with pain and swelling of the wrist. In severe cases there is constitutional

illness, with pyrexia. *On examination* the wrist is swollen, most noticeably at the dorsum; the swelling is caused partly by fluid within the joint and partly by synovial thickening. The overlying skin is warmer than normal and it is often reddened. All movements of the wrist are impaired, usually markedly, and they are painful. *Radiographs* in the early stages show no alteration from the normal. Later, if the infection persists, there is diffuse osteoporosis, with loss of cartilage space and possibly some destruction of bone (Fig. 17, p. 33). *Investigations:* The blood shows a raised sedimentation rate and a polymorphonuclear leucocytosis. The causative organism can usually be isolated from the joint fluid.

Treatment. Appropriate chemotherapy is begun immediately. The wrist is immobilised on a splint. The joint is aspirated or drained by incision, and the appropriate antibiotic drug is injected into it. When the infection has been overcome active wrist movements are encouraged.

PYOGENIC ARTHRITIS OF THE JOINTS OF THE HAND

Any of the small joints of the hand may be infected by pyogenic organisms. The distal interphalangeal joints of the digits are prone to infection spreading from a suppurative lesion in the adjacent pulp space.

Clinical features. The affected joint is swollen, hot, and red. Movements are markedly impaired. *Radiographs* show no early change, but later there are osteoporosis and diminution of the cartilage space (Fig. 166).

Treatment. Reliance is placed upon chemotherapy, drainage of the joint if suppuration occurs, and immobilisation during the acute stage of infection. But splintage must be discontinued and active exercises begun as soon as the infection subsides.

Fig. 166

Fig 166
Pyogenic arthritis with destruction of interphalangeal joint of thumb.

RHEUMATOID ARTHRITIS OF THE WRIST

(General description of rheumatoid arthritis, p. 34.)

One or both wrists are frequently affected in rheumatoid arthritis, usually in company with several other joints.

Clinical features. The clinical features are like those of rheumatoid arthritis in other joints. The symptoms are pain in the wrist, swelling, stiffness, and weakness of grip. *On examination* the wrist is swollen from synovial thickening. The overlying skin is warmer than normal. All movements are limited, and painful at the extremes. *Radiographs* show no abnormality at first. Later, there is diffuse osteoporosis in the area of the joint. Eventually, in progressive disease, destruction of cartilage leads to narrowing of the joint space (Fig. 168). *Investigations:* The erythrocyte sedimentation rate is increased during the active phase of the disease.

Course. As in other joints, there is a tendency for the disease to become quiescent after months or years of activity. The wrist is left more or less damaged. Secondary degenerative (osteoarthritic) changes may cause continued pain even after the active rheumatoid changes have abated

Treatment. The general principles of treatment, as already described, apply equally to the wrist. There, as in other joints, the treatment depends upon the severity of the inflammatory reaction When the reaction is severe, temporary splintage in plaster is sometimes required When it is slight or moderate, active use of the wrist is encouraged. Physiotherapy, in the form of hot wax baths or short-wave diathermy, followed by active exercises, is of definite value Drugs such as aspirin and phenylbutazone should be tried The advisability of systemic treatment by gold salts, artificial pyrexia, or cortisone, must be assessed for each individual case, according to the extent and severity of the arthritis

Operative treatment. If degenerative changes lead to persistent disabling pain arthrodesis of the wrist may be required.

RHEUMATOID ARTHRITIS OF THE JOINTS OF THE HAND

The joints of the hands—especially the metacarpo-phalangeal joints—are commonly affected in rheumatoid arthritis.

Clinical features. Usually many or all of the hand joints are

affected, but sometimes a single joint is involved. There are pain and swelling of the joints, with a feeling of stiffness and, eventually, obvious deformity of the hand (Fig. 167). *On examination* the joints are swollen and the overlying skin is warmer than normal. Movements are moderately restricted. A striking

Fig. 167

Typical appearance of hand in long-established rheumatoid arthritis of the metacarpo-phalangeal and interphalangeal joints

Fig. 168

Rheumatoid arthritis of the wrist and certain interdigital joints Note the osteoporosis, loss of cartilage space and, in places, erosion of bone.

and constant feature in the later stages is ulnar deviation of the fingers at the metacarpo-phalangeal joints

Treatment. The general plan of treatment is that for rheumatoid arthritis as a whole. In the hand, however, the joints must not be immobilised, even during the most acute stage of the disease. Active use of the hand and fingers must be encouraged. Hot wax baths and supervised exercises are helpful.

TUBERCULOUS ARTHRITIS OF THE WRIST

(General description of tuberculous arthritis, p. 37.)

Tuberculosis is seldom encountered in the wrist. When it does occur, it is often in patients who are old and frail, or in

those with multiple foci of tuberculosis elsewhere in the body. The features of the disease are like those of tuberculous arthritis at other sites

Treatment. In most cases treatment is along the lines suggested for tuberculosis of other major joints.

In the aged, resistance to tuberculosis sometimes seems to be lost and the disease progresses despite treatment, usually with a profusely discharging sinus In such a case the hand is rendered useless and amputation may have to be considered.

OSTEOARTHRITIS OF THE WRIST

(General description of osteoarthritis, p. 41.)

The degenerative changes of osteoarthritis are rather common in the wrist because of the frequency of injury to the joint and the proclivity of scaphoid fractures to non-union.

Cause. Although it is essentially a wear-and-tear process osteoarthritis seldom develops in a wrist that was previously normal. The wear-and-tear is nearly always accelerated by previous injury to, or disease of, the joint surfaces The commonest predisposing factors are fracture of the scaphoid (especially when the fracture fails to unite), dislocation of the lunate bone, Kienbock's disease of the lunate bone, and " burnt out " rheumatoid arthritis.

Pathology. The predominant change is degeneration and wearing away of the articular cartilage lining the joint surfaces. The changes eventually involve all the carpal joints as well as the radio-carpal joint.

Clinical features. Months or years after one of the predisposing conditions mentioned, the patient notices gradually increasing pain and stiffness of the wrist, worse on activity. *On examination* the wrist is slightly thickened from bony irregularity but the swelling is not marked The skin temperature is normal. Movements are limited, and painful if forced at the extremes. *Radiographs* show narrowing of the cartilage space and sharpening or spurring of the bone at the joint margins The causative condition (for example, an ununited fracture of the scaphoid) is frequently evident (Fig. 169).

Treatment. In mild cases the condition is best left alone, especially if the patient can avoid subjecting the wrist to heavy stress. When active treatment seems necessary, a choice must be made between conservative and operative methods. Conservative treatment can only diminish the symptoms; it can never remove them. Nevertheless it is usually worth a trial. The most useful method is to combine physiotherapy, in the form of short-wave diathermy, with the provision of a firm wrist support of moulded leather or plastic.

Operative treatment sometimes becomes necessary when the disability is severe. The only reliable method is by total arthrodesis of the wrist, ablating the radiocarpal and all the intercarpal joints.

FIG. 169
Osteoarthritis of the wrist caused by an ununited fracture of the scaphoid bone.

OSTEOARTHRITIS OF THE JOINTS OF THE HAND

The metacarpo-phalangeal joints and the interphalangeal joints of the hand are frequently the site of osteoarthritis in the elderly. Such manifestations are relatively unimportant and in most cases treatment is not required. A special example demands further consideration—namely, osteoarthritis of the trapezio-metacarpal joint (carpo-metacarpal joint of the thumb).

OSTEOARTHRITIS OF THE TRAPEZIO-METACARPAL JOINT

This is a common affection in elderly women and it often impairs seriously the function of the thumb.

Clinical features. Women past middle age are most commonly affected. There is pain, localised to the trapezio-metacarpal joint, on using the thumb. The disability slowly increases over the years until activities like sewing or darning become virtually

those with multiple foci of tuberculosis elsewhere in the body. The features of the disease are like those of tuberculous arthritis at other sites.

Treatment. In most cases treatment is along the lines suggested for tuberculosis of other major joints.

In the aged, resistance to tuberculosis sometimes seems to be lost and the disease progresses despite treatment, usually with a profusely discharging sinus. In such a case the hand is rendered useless and amputation may have to be considered.

OSTEOARTHRITIS OF THE WRIST

(General description of osteoarthritis, p. 41.)

The degenerative changes of osteoarthritis are rather common in the wrist because of the frequency of injury to the joint and the proclivity of scaphoid fractures to non-union.

Cause. Although it is essentially a wear-and-tear process osteoarthritis seldom develops in a wrist that was previously normal. The wear-and-tear is nearly always accelerated by previous injury to, or disease of, the joint surfaces. The commonest predisposing factors are fracture of the scaphoid (especially when the fracture fails to unite), dislocation of the lunate bone, Kienbock's disease of the lunate bone, and "burnt out" rheumatoid arthritis.

Pathology. The predominant change is degeneration and wearing away of the articular cartilage lining the joint surfaces. The changes eventually involve all the carpal joints as well as the radio-carpal joint.

Clinical features. Months or years after one of the predisposing conditions mentioned, the patient notices gradually increasing pain and stiffness of the wrist, worse on activity. *On examination* the wrist is slightly thickened from bony irregularity but the swelling is not marked. The skin temperature is normal. Movements are limited, and painful if forced at the extremes. *Radiographs* show narrowing of the cartilage space and sharpening or spurring of the bone at the joint margins. The causative condition (for example, an ununited fracture of the scaphoid) is frequently evident (Fig. 169).

Pathology. The condition is similar to, but probably not identical with, osteochondritis of developing epiphysial centres in children (p. 77), such as Perthes' disease. It is more likely that it is simply an example of avascular necrosis. The bone becomes granular in texture, small dense fragments being interspersed with softened

FIG. 171
Kienbock's disease of the lunate bone Note the increased density, fragmentation, and beginning compression of the bone

areas. In this state the bone crumbles easily, and under the pressure imposed by muscle action and use of the wrist it gradually becomes squashed into a thin saucer-shaped mass. The overlying cartilage dies. After about two years the bone texture is restored to normal, but the bone remains deformed and is *devoid of a* smooth cartilaginous covering. The bone behaves like a piece of grit in a bearing and leads gradually to the development of osteoarthritis of the wrist.

Clinical features. There is pain in the wrist, most marked at the centre of the joint over the lunate area. The *pain is* worse during active use of the wrist. Because of the pain, the strength of grip is impaired. *On examination* there is discomfort on pressure over the lunate bone. Movements of the wrist are limited and cause pain if forced. *Radiographs* are diagnostic. In the early stages the lunate appears slightly more dense than the surrounding

impossible. *On examination* the trapezio-metacarpal joint is prominent and slightly thickened. Active or passive movements of the thumb metacarpal cause pain. The range of movement at this joint varies widely even in normal individuals, so its measurement is of little practical value. *Radiographs* show narrowing of the cartilage space and sharpening or "spurring" of bone at the joint margins (Fig. 170). In many cases the joint is subluxated.

FIG 170
Osteoarthritis of the trapezio-metacarpal joint.

Treatment. In the early stages the condition is best left alone. For moderate symptoms a course of short-wave diathermy may be tried. If the symptoms become disabling operation is advisable. The choice lies between arthroplasty and arthrodesis. Arthroplasty is done simply by excising the trapezium, allowing the resulting gap to fill with fibrous tissue. It gives results that are quite adequate for the usual elderly sufferer from this disorder. But if heavy use is to be demanded of the hand (as in the case of a labourer, for example), arthrodesis of the trapezio-metacarpal joint is to be preferred.

KIENBÖCK'S DISEASE

(Osteochondritis of the lunate bone)

Kienbock's disease is an affection of the lunate bone characterised by temporary softening, fragmentation, and liability to deformation. It tends to give rise, later, to osteoarthritis of the wrist.

Cause. The precise cause is unknown, but a disturbance of blood supply, possibly from thrombosis of a nutrient vessel, is believed to be responsible. Repeated injury (for example, using the front of the wrist to drive a chisel in carpentry) is probably a predisposing factor.

effective treatment, infection may spread to adjacent tissue planes; occasionally it may give rise to spreading lymphangitis or to septicaemia.

Surgical anatomy. A knowledge of the anatomy of the fascial

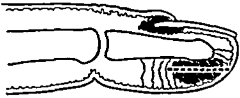

FIG 172

Diagrammatic section showing the site of suppuration in pulp-space infection. Note the partition of the pulp by numerous vertical fibrous strands. To drain the space these must be incised transversely (interrupted line). The diagram also depicts a paronychial infection, with extension under the nail. To drain this adequately the base of the nail would have to be removed.

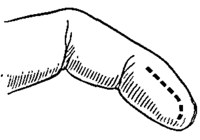

FIG 173

FIG 174

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proximal half of the nail is removed.

spaces [1] of the hand is indispensable for the correct treatment of hand infections.

The nail fold and subungual space The plane beneath the nail fold is potentially continuous, at the base and sides of the nail,

[1] The term "space" as used in this connection is a misnomer. It refers to the interval or plane between adjacent tissues and, in the normal hand, it is only a potential space.

bones, and if its depth is compared with that of the lunate bone of the sound wrist it is seen to be reduced, though only slightly at first (Fig. 171). Later, the bone has a fragmented appearance, small areas of increased density being scattered through it, and the flattening of the bone becomes obvious. Later still, signs of osteoarthritis of the wrist joint are evident.

Treatment. This depends upon the duration of the symptoms and the degree of damage to the wrist. If the condition is diagnosed within a few months of the onset, before the wrist joint is severely damaged, the lunate bone should be excised. The gap fills with fibrous tissue and the function of the wrist, though far from normal, is reasonably good. When symptoms have been present for many months osteoarthritis is usually already present. At that stage excision of the lunate is of no avail. Treatment should be the same as for osteoarthritis of the wrist (p. 254).

EXTRA-ARTICULAR DISORDERS ABOUT THE WRIST AND HAND

ACUTE INFECTIONS OF THE FASCIAL SPACES OF THE HAND

Acute infections of the hand account for a considerable proportion of the work of a casualty department and are of great importance in industrial medicine. Unless they are treated correctly and efficiently they can lead to prolonged or even permanent disability, with impairment of working capacity.

Classification. Excluding minor superficial infections, there are six types to be considered: 1) nail-fold infection (paronychia); 2) pulp-space infection (whitlow, felon); 3) other subcutaneous infections; 4) thenar space infection; 5) mid-palmar space infection; 6) tendon-sheath infection.

Cause. All types are caused by infection with pyogenic bacteria. The commonest causative organism is the staphylococcus; the next most common is the streptococcus. Minor injury such as a prick or abrasion is important etiologically.

Pathology. The organisms reach the tissue planes by direct implantation from outside, often as the result of a trivial injury such as a prick or abrasion. They set up an acute inflammatory reaction which in many cases goes on to suppuration Without

and the first and second lumbrical muscles in front. Medially it is separated from the mid-palmar space by a fibrous septum that extends deeply from the fascia on the deep surface of the flexor tendons to the fascia covering the interossei and adductor pollicis muscle (Fig. 175). The space is prolonged forwards into the

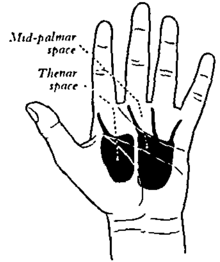

FIG. 176

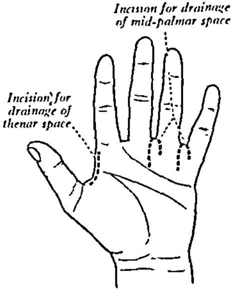

FIG. 177

Figure 176—Surface marking of the thenar and mid-palmar spaces. The thenar space is continuous with the first lumbrical canal and the mid-palmar space with the second, third, and fourth lumbrical canals. Figure 177—Incisions for drainage.

delicate sheath that surrounds the first lumbrical muscles. It sometimes communicates also with the second lumbrical canal.

The mid-palmar space. This lies under the medial (ulnar) half of the hollow of the palm. It is the interval between the interossei and metacarpal bones behind and the flexor tendons (in their sheaths) of the middle, ring, and little fingers in front (Fig. 175). Laterally, it is separated from the thenar space by the fibrous septum already described. The space is prolonged forwards into the sheaths of the second, third, and fourth lumbrical muscles.

The flexor tendon sheaths. Distinction must be made between the tough fibrous sheaths, which exist only in the digits, and the flimsy synovial sheaths, which line the fibrous sheaths and, in the case of the thumb and little finger, extend proximally into the palm.

with the subungual space deep to the nail. Infection beginning in the nail fold may therefore easily spread under the nail (Fig. 172), and the resulting abscess cannot be drained effectively unless part of the nail is removed.

The pulp space. The interval between the front of the distal phalanx and the skin is traversed by tough fibrous partitions, which subdivide the space into numerous fat-filled cells (like the cells of a honeycomb) disposed at right angles to the skin surface (Fig. 172). Effective drainage of the space can be secured only by an incision that cuts across the axis of these fibrous partitions.

Other subcutaneous spaces. The nail fold and the pulp space are strictly subcutaneous spaces—that is, they lie immediately deep to the skin—but they have special features which have demanded separate consideration. Infections may occur in the subcutaneous plane at any point in the hand: *common sites are the middle* segment of the fingers, the palm of the hand, and the dorsum of the hand or fingers. Infections of this plane are to be distinguished from those of the deeper spaces described below.

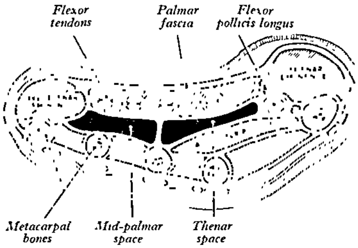

FIG. 175

The thenar space and the mid-palmar space shown in diagrammatic transverse section

The thenar space This lies deeply under the lateral (radial) half of the hollow of the palm It is the interval between the adductor pollicis muscle behind and the flexor tendon of the index finger

Principles of treatment. Before suppuration has occurred, the aim of treatment is to abort the infection and avoid the necessity for operation. The hand is rested in a sling and penicillin is given systemically by injection. Relatively few cases are seen at a stage early enough to permit success from this expectant treatment.

When suppuration has already occurred, as indicated by severe throbbing pain, intense local tenderness, pyrexia, and loss of function, surgical drainage of the abscess is required. There should be no undue delay, but it is often advantageous to combat the constitutional disturbance by penicillin for a few hours before operation is undertaken. After adequate drainage has been secured the wound is packed lightly open with vaselined gauze for two days. Thereafter, dry dressings are used and active finger exercises are encouraged.

SPECIAL FEATURES OF INDIVIDUAL LESIONS

NAIL-FOLD INFECTION (paronychia). *Clinical features :* There are pain, redness, and swelling at one or both sides of the nail fold and at the base of the nail. There is local tenderness over the reddened area. If suppuration has extended deep to the nail there is marked tenderness on pressure upon the nail. *Complications :* These are : 1) extension to the pulp space ; and 2) chronic paronychia, following inadequate treatment of the acute infection. *Treatment :* In this type of infection conservative measures are often successful if begun within a few hours of the onset. When drainage is required, the skin over the base of the nail is raised as a flap, and the proximal one-half of the nail is removed (Fig. 174).

PULP-SPACE INFECTION (whitlow). *Clinical features :* The pulp is markedly swollen, red, and very tender. Severe throbbing pain suggests that suppuration is present. *Complications :* These are : 1) osteomyelitis of the terminal phalanx, often leading to necrosis and sequestration of its distal half ; 2) pyogenic arthritis of the distal interphalangeal joint ; and 3) spread of infection to the flexor tendon sheath (suppurative tenosynovitis). *Treatment :* Conservative measures are seldom successful except in the earliest stages. Surgical drainage is by a lateral incision just in front of the plane of the terminal phalanx (Fig. 173) ; it is deepened transversely across the pulp of the finger but should not extend proximally beyond a point a quarter of an inch distal to the terminal skin crease lest the flexor tendon sheath be inadvertently opened.

SUBCUTANEOUS INFECTIONS (other than nail-fold and pulp-space infections). *Clinical features :* The infection may arise in any part of the hand or fingers. There is a localised swelling with redness and

In acute infections of the tendon sheaths (acute infective tenosynovitis) the pus is within the synovial sheath and it is confined only by the limits of the sheath. The flexor sheaths of the index, middle, and ring fingers end proximally at the level of the transverse palmar skin crease (Fig. 178). The sheaths of the thumb and little finger extend proximally through the palm to end one inch above the level of the wrist joint. The proximal part of the sheath for the thumb is known as the radial bursa. The sheath for the little

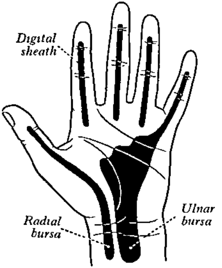

FIG 178

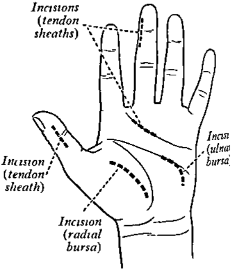

FIG 179

Figure 178—The synovial flexor sheaths Whereas the sheaths for the index, middle, and ring fingers end proximally at the bases of the fingers those for the thumb and little finger extend upwards to become continuous with the radial bursa and the ulnar bursa respectively. Figure 179—Incisions for drainage and irrigation of tendon sheaths and bursae.

finger opens out proximally into the ulnar bursa, which encloses the grouped tendons of flexor digitorum sublimis and flexor digitorum profundus (Fig 178).

Clinical features. In general, the symptoms of acute hand infections are local pain, swelling, and loss of function. There is often some degree of constitutional disturbance, with pyrexia *On examination* there are obvious swelling, redness of the skin (except in deep infections), and marked local tenderness over the site of the infection. Special features of the individual lesions are given below.

a similar condition complicates rheumatoid arthritis without any demonstrable bacterial infection.

Pathology. The flexor tendon sheaths in the lower forearm and hand are those most commonly affected; less often the lesion is confined to the extensor sheaths. The affected sheaths are greatly thickened and show the changes of chronic inflammation. In

FIG. 180
Compound palmar ganglion The swellings in the palm and at the front of the wrist are continuous deep to the flexor retinaculum.

most cases there is histological evidence of tuberculosis. The sheaths often contain an excess of fluid and there may be collections of small fibrinous bodies. The tendons themselves are affected only slightly

Clinical features. There is a gradual onset of swelling, with mild aching pain, in the region of the affected tendon sheaths—usually the flexor sheaths of the lower forearm and hand. The function of the fingers and thumb is impaired. *On examination* the swelling is confined to the line of the tendon sheaths. Characteristically it affects the lower two or three inches of the front of the forearm and the proximal part of the palm (Fig. 180). Sometimes the flexor sheaths of the fingers and thumb are also swollen, giving the digits a fusiform appearance. In many cases fluctuation can be elicited between the forearm swelling and the swelling in the palm This clinical sign depends upon the presence of fluid within the tendon sheaths, and it is by no means always found. The fully developed condition, with swelling in forearm and

tenderness. In many cases the infection has spread through the skin from a subcuticular infection. Care must be taken not to confuse subcutaneous infections with the deeper space infections. *Complications:* These are 1) sloughing of skin over the lesion; and 2) spread to the deep spaces or to the flexor tendon sheaths. *Treatment:* Surgical drainage is by a short incision appropriately placed to reach the abscess without harming important structures or leaving an awkward scar.

THENAR-SPACE INFECTION. This is an uncommon lesion. It usually arises by extension from a subcutaneous lesion or a tendon-sheath infection. *Clinical features:* The radial half of the palm is ballooned out and the swelling extends to the dorsal aspect of the web between thumb and index finger. *Treatment:* Drainage is by an incision at the dorsal aspect of the first web space (Fig. 177).

MID-PALMAR SPACE INFECTION. This is an uncommon lesion. It usually arises by extension from a subcutaneous lesion or a tendon-sheath infection. *Clinical features:* The ulnar half of the palm is ballooned out. Movements of the fingers are restricted and painful. *Treatment:* Drainage is by a web-splitting incision between the middle and ring fingers or between the ring and little fingers (Fig. 177).

TENDON-SHEATH INFECTION. This is uncommon, but important because prompt treatment is essential if the function of the finger is to be preserved. *Clinical features:* The finger is swollen throughout its length, and acutely tender over the flexor tendon sheath. It is held semi-flexed and the patient is unwilling to extend it because of pain. *Complications:* These are: 1) necrosis of the tendons and adhesions between tendon and sheath, causing permanent stiffness of the finger in semi-flexion, and 2) spread of infection to involve the radial bursa (from the flexor sheath of the thumb), or the ulnar bursa (from the sheath of the little finger). *Treatment:* Systemic penicillin is begun immediately. The sheath is opened at its proximal end in the palm and at its distal end (Fig. 179), and irrigated with penicillin solution through a fine tube passed along the sheath, the tube is withdrawn and the wound packed lightly open.

If the radial bursa or the ulnar bursa is infected it must be drained and irrigated through an additional incision in the palm (Fig. 179).

CHRONIC INFECTIVE TENOSYNOVITIS

(Including compound palmar ganglion)

Chronic inflammation of tendon sheaths in the lower forearm and hand is usually a response to low-grade infection. It is entirely distinct from acute tenosynovitis and is not preceded by it.

Cause. In most cases it is caused by infection with the tubercle bacillus. Sometimes other organisms are responsible. Occasionally

a similar condition complicates rheumatoid arthritis without any demonstrable bacterial infection.

Pathology. The flexor tendon sheaths in the lower forearm and hand are those most commonly affected; less often the lesion is confined to the extensor sheaths. The affected sheaths are greatly thickened and show the changes of chronic inflammation. In

FIG. 180

Compound palmar ganglion The swellings in the palm and at the front of the wrist are continuous deep to the flexor retinaculum.

most cases there is histological evidence of tuberculosis. The sheaths often contain an excess of fluid and there may be collections of small fibrinous bodies. The tendons themselves are affected only slightly.

Clinical features. There is a gradual onset of swelling, with mild aching pain, in the region of the affected tendon sheaths—usually the flexor sheaths of the lower forearm and hand. The function of the fingers and thumb is impaired. *On examination* the swelling is confined to the line of the tendon sheaths. Characteristically it affects the lower two or three inches of the front of the forearm and the proximal part of the palm (Fig. 180). Sometimes the flexor sheaths of the fingers and thumb are also swollen, giving the digits a fusiform appearance. In many cases fluctuation can be elicited between the forearm swelling and the swelling in the palm. This clinical sign depends upon the presence of fluid within the tendon sheaths, and it is by no means always found. The fully developed condition, with swelling in forearm and

palm and fluctuation between the two, constitutes *compound palmar ganglion.*

At first the range of movements of the fingers and thumb is impaired only slightly, if at all. Later, there is moderate restriction of flexion and extension of the digits, with corresponding loss of function. A tuberculous lesion may be discovered elsewhere in the body.

Diagnosis. A persistent swelling of gradual onset in the line of the tendon sheaths in the lower forearm and hand always suggests chronic tenosynovitis. Fluctuation between the forearm swelling and the palmar swelling provides strong corroborative evidence. If an active tuberculous lesion is found elsewhere in the body it is reasonable to infer that the tenosynovitis is also tuberculous

Treatment. In mild cases in which the function of the fingers and thumb is not impaired conservative treatment is advised. The wrist and forearm are immobilised in plaster of Paris for three months, the fingers being left free.

In most cases, however, operation is recommended It consists in excising thoroughly all the thickened and oedematous tendon sheaths. After operation finger movements are encouraged and practised assiduously under the supervision of a physiotherapist. In tuberculous cases a course of the appropriate antibiotic drugs is given.

BONE TUMOURS IN THE HAND

(General description of bone tumours, p. 66.)

The only bone tumour that requires special mention here is the benign chondroma.

CHONDROMA

A chondroma or benign cartilage tumour occurs in two forms: *enchondroma*, which grows within the bone and expands it; and *ecchondroma*, which grows mainly outwards from the surface of the bone Both types are prone to occur in the metacarpals and phalanges of the hand. The tumours are often multiple, and they may cause ugly swelling and deformity of the fingers (Fig. 64, p 85).

Treatment. If small, the tumour should be treated expectantly. Operation is required only if it is found to be enlarging. Large tumours should be excised, the bone substance being restored, if necessary, by grafts of cancellous bone.

SOFT-TISSUE TUMOURS IN THE HAND

(General description of soft-tissue tumours, p. 108.)

Special mention must be made of an unusual tumour that is seldom encountered outside the hand—namely, the giant-cell tumour of tendon sheath.

Giant-cell Tumour of Tendon Sheath

This is a benign tumour, but it sometimes recurs locally unless it is removed entire. It arises from the sheath of a tendon, or from the fibrous expansion of an extensor tendon in a finger. As it enlarges it burrows between the tissue planes, taking the line of least resistance. Eventually it may form a bulky mass which almost surrounds the finger like a collar. On section the tumour is fleshy; histologically it is composed of cells of many forms, including giant cells of foreign-body type, xanthoma cells containing cholesterol, and fibroblasts.

Treatment. The tumour should be excised entire.

COMPRESSION OF THE MEDIAN NERVE IN THE CARPAL TUNNEL

(Carpal tunnel syndrome)

This disorder, described only within the last ten years, is now recognised as a common cause of discomfort in the hand, especially in elderly women. Its essential feature, as the name implies, is a constriction of the median nerve behind the flexor retinaculum.

Cause. Any space-occupying lesion within the carpal tunnel may be responsible The commonest are chronic inflammatory thickening of the tendon sheaths and osteoarthritis of the wrist. In many cases no primary cause can be discovered.

Pathology. The median nerve lies beneath the flexor retinaculum in company with the flexor tendons of the hand. If the available space within this strong-walled tunnel is reduced the nerve is compressed against the flexor retinaculum. When the retinaculum is divided in such a case the nerve is seen to be constricted where it lay behind it. The ulnar nerve does not pass behind the flexor retinaculum, so it is not liable to compression in this way.

Clinical features. The condition is commonest in women past middle age. The symptoms are sensory and motor. There is

tingling, numbness, or discomfort in the radial three and a half digits (that is, in the median distribution), and there is a feeling of clumsiness in carrying out fine movements such as those concerned in sewing. *On examination* the findings vary with the degree and duration of the compression. At first there are no objective findings Later there is blunting of sensation in the median distribution. Eventually there are also wasting and weakness of the median-innervated small muscles of the hand

Diagnosis. Care must be taken to exclude other causes of neurological disturbance in the hand, especially those arising in the neck from interference with the brachial plexus, and lesions of the median nerve elsewhere in its course. Other neurological disorders such as progressive muscular atrophy and syringomyelia must also be considered.

Treatment. The flexor retinaculum is divided to decompress the nerve.

GANGLION

(Simple ganglion)

A ganglion is the commonest cystic swelling at the back of the wrist.

Pathology. Conflicting views have been put forward on the origin of ganglia. Some believe that they represent a degenerative process Others claim that they are benign tumours of tendon sheath or joint capsule The cyst wall is of fibrous tissue and there is no true endothelial lining It is connected at some point with a joint capsule or tendon sheath, but there is no communication between the joint cavity or tendon sheath and the interior of the cyst The cyst may be unilocular or multilocular. The contained fluid is clear and viscous

Clinical features. Ganglia are commonest at the back of the wrist, where they are often seen in adults of any age (Fig. 181). They also occur, less commonly, in the palm and fingers Ordinarily there are no symptoms other than the swelling itself and, sometimes, discomfort or slight pain *On examination* the swelling may be soft and obviously cystic, but more often it is tense. It is often mistaken for a bony prominence; but careful tests will show that it is fluctuant.

Complications. A ganglion arising deeply in the wrist or palm may interfere mechanically with the ulnar or the median nerve.

There will be motor and usually sensory impairment in the distribution of the particular branch affected.

FIG. 181
A simple ganglion at the back of the wrist. This is the commonest site.

Treatment. A ganglion is harmless and in the absence of pain or complications it can safely be left alone. If treatment is required excision should usually be advised. Occasionally the ganglion can be dispersed subcutaneously by firm local pressure. This treatment does no harm, but the ganglion will slowly reappear.

A ganglion that is interfering with a peripheral nerve demands operative excision without delay.

DUPUYTREN'S CONTRACTURE

(Contracture of the palmar aponeurosis)

This is an easily recognised condition characterised by flexion contracture of one or more of the fingers from thickening and shortening of the palmar aponeurosis.

Cause. This is unknown. There is believed to be a hereditary predisposition. Injury possibly plays a part but its exact significance is uncertain.

Pathology. The palmar aponeurosis (palmar fascia) is normally a thin but tough membrane whose fibres radiate from the termination of the palmaris longus tendon at the front of the wrist to gain insertion into the proximal and middle phalanges of

the fingers. It lies immediately beneath the skin. In Dupuytren's contracture the aponeurosis, or part of it, becomes greatly thickened (often to a quarter of an inch or more), and it contracts, drawing the fingers into flexion at the metacarpo-phalangeal and

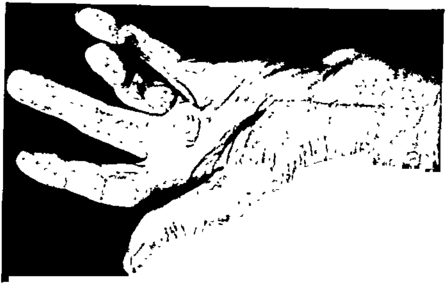

FIG 182
A typical example of Dupuytren's contracture of the palmar aponeurosis

proximal interphalangeal joints. The medial half of the aponeurosis is affected most, and serious flexion deformity is usually confined to the ring and little fingers The joints themselves are unaffected at first, but in long-established cases secondary capsular contractures occur.

Clinical features. The affection is much more common in men than in women It is commonly bilateral. The earliest sign is a small thickened nodule in the mid-palm opposite the base of the ring finger The area of thickening gradually spreads from this point, giving rise eventually to firm cord-like bands that extend into the ring or little finger, or both, and prevent full extension of the metacarpo-phalangeal and proximal interphalangeal joints (Fig 182). The skin is closely adherent to the fascial bands, and is often puckered. The flexion deformity becomes progressively worse in the course of months or years.

Treatment. The only effective treatment is by operation. That does not imply, however, that operation is necessary in every case :

a contracture that is not progressing rapidly is often better left alone, especially in an elderly patient. Operation entails excision of the thickened part of the palmar fascia by painstaking dissection. Simple transverse division of the taut contracted bands is to be condemned, for the contracture will quickly recur.

RUPTURE OR SEVERANCE OF TENDONS IN THE HAND

Most tendon divisions in the hand result from cuts with sharp objects such as glass or knives. Certain tendons are prone to rupture: thus the extensor tendon of a finger is easily torn from

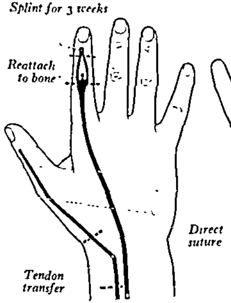

FIG. 183

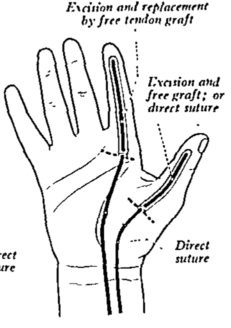

FIG. 184

Treatment of tendon injuries at various sites Figure 183—Extensor tendons. Figure 184—Flexor tendons For details, see text.

its insertion into the distal phalanx by sudden forced flexion of the finger; and the extensor pollicis longus is liable to spontaneous rupture after fractures of the lower end of the radius in consequence of its becoming frayed where it crosses the roughened bone.

Clinical features and diagnosis. Loss of function of a tendon is obvious clinically. When the findings are correlated with the history the diagnosis is usually clear.

the fingers. It lies immediately beneath the skin. In Dupuytren's contracture the aponeurosis, or part of it, becomes greatly thickened (often to a quarter of an inch or more), and it contracts, drawing the fingers into flexion at the metacarpo-phalangeal and

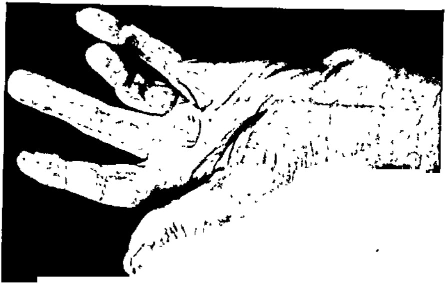

FIG. 182
A typical example of Dupuytren's contracture of the palmar aponeurosis.

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Clinical features. The affection is much more common in men than in women. It is commonly bilateral The earliest sign is a small thickened nodule in the mid-palm opposite the base of the ring finger. The area of thickening gradually spreads from this point, giving rise eventually to firm cord-like bands that extend into the ring or little finger, or both, and prevent full extension of the metacarpo-phalangeal and proximal interphalangeal joints (Fig. 182). The skin is closely adherent to the fascial bands, and is often puckered. The flexion deformity becomes progressively worse in the course of months or years.

Treatment. The only effective treatment is by operation That does not imply, however, that operation is necessary in every case :

SEVERANCE OF EXTENSOR TENDONS AT THE BACK OF THE HAND. This injury has a good prognosis. There is a tendency to spontaneous union with recovery of normal function. *Treatment :* Primary suture should be undertaken if the case is seen fresh. Failing this, expectant treatment may be adopted for two or three months, during which spontaneous restoration of function can be hoped for. If disability persists, freshening and direct suture of the divided ends is advised.

RUPTURE OF EXTENSOR POLLICIS LONGUS TENDON complicating fracture of lower end of radius. The tendon gives way after becoming frayed

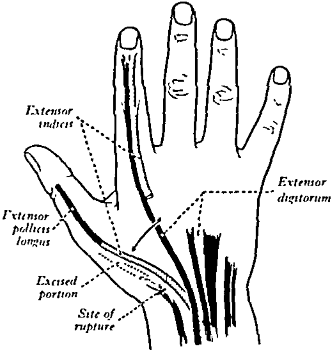

FIG. 186

Transfer of extensor indicis to replace a ruptured extensor pollicis longus. This transfer is to be preferred to direct suture when the ends of the ruptured tendon are frayed.

by repeated movement over the roughened lower end of the radius. The extensive fraying makes direct suture unsatisfactory. *Treatment :* The tendon of extensor indicis is divided at the level of the neck of the second metacarpal, re-routed towards the thumb, and sutured to the freshened distal stump of the extensor pollicis longus (Fig. 186).

INJURIES OF FLEXOR TENDONS

DIVISION WITHIN A FIBROUS FLEXOR SHEATH OF A FINGER. Severance at this site presents the most difficult problem of all tendon injuries. The

Treatment. This varies according to the tendon affected and the site of severance (see below and Figs. 183-184). Sometimes treatment is unnecessary or undesirable, but more often operative reconstruction is to be advised: the tendon is either sutured directly or replaced by a free tendon graft or by a tendon transfer, according to circumstances.

SPECIAL FEATURES OF INDIVIDUAL LESIONS

Injuries of Extensor Tendons

Avulsion of extensor tendon from its insertion into the distal phalanx of a finger. This is known as "mallet finger." It is caused by sudden forced flexion of the distal interphalangeal joint—for instance, by a blow on the tip of the finger from a cricket ball. In a few cases a small fragment of bone is avulsed with the tendon. The patient is unable fully to extend the distal interphalangeal joint (Fig. 185). *Treatment:* Immediate treatment is to splint the finger for three weeks with the

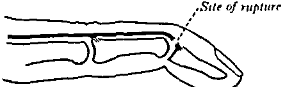

Fig. 185

Mallet finger. The extensor tendon is avulsed from its insertion. The terminal joint cannot be extended fully

distal interphalangeal joint fully extended and the proximal interphalangeal joint flexed 90 degrees—a position which ensures that the distal part of the extensor expansion is relaxed. The avulsed tendon always unites to the bone, but often with lengthening, in which case the deformity persists. The choice then lies between accepting the disability, which is slight, or operation. Operation entails shortening the extensor tendon by excision of a short section at the level of the middle phalanx.

Rupture of the middle slip of the extensor expansion. This is caused by sudden forced flexion of the proximal interphalangeal joint, the middle slip of the extensor expansion being torn from its attachment to the middle phalanx. The patient is unable to extend the proximal interphalangeal joint fully. The distal joint becomes hyperextended. *Treatment:* The tendon is reattached by wire sutures.

Technique: Direct suture is usually unsatisfactory because the tendon sticks to the narrow sheath at the site of suture. The most satisfactory method is to remove the flexor sublimis tendon entire (to make more room in the sheath) and to replace the whole of the digital part of the flexor profundus tendon by a free tendon graft (from a toe extensor) sutured proximally to the profundus tendon in the palm, and inserted distally into a drill hole in the distal phalanx (Fig. 187). This method eliminates the need for a tendon junction within the sheath.

Division of flexor pollicis longus in the thumb. The problem is less difficult than that presented by division of both flexor tendons in a finger. Repair may be attempted either by direct suture, or by replacement of the digital part of the tendon by a free graft as described for the fingers.

Divisions of flexor tendons in the palm or wrist. Direct suture is advised. It may be done primarily if the wound is clean. The prognosis is good if a single tendon is affected but it is uncertain in cases of multiple tendon divisions at the front of the wrist, especially if the nerves are also injured.

DE QUERVAIN'S TENOVAGINITIS

(Tenovaginitis of the abductor pollicis longus and extensor pollicis brevis)

This is a common and well recognised condition characterised by pain over the tip of the radial styloid process and a palpable nodule in the course of the abductor pollicis longus and extensor pollicis brevis tendons.

Cause. The precise cause is unknown. The excessive friction of over-use may be a factor, for the condition often appears to follow oft-repeated actions like wringing clothes.

Pathology. The fibrous sheaths of the abductor pollicis longus and the extensor pollicis brevis are thickened where they cross the tip of the radial styloid process. The tendons themselves appear normal. The condition is possibly analogous to that other common form of tenovaginitis, " trigger " finger.

Clinical features. The condition is commonest in middle-aged women. The main symptom is pain on using the hand, especially when movement tenses the abductor pollicis longus and extensor pollicis brevis tendons (as in lifting a teapot). *On examination* there is well marked local tenderness at the point where the tendons cross the radial styloid process (Fig. 188), and the thickened fibrous

results of operative repair are unpredictable, and in many cases the operation fails to restore a useful range of active finger movement.

Treatment. If a flexor sublimis tendon alone is divided and the flexor profundus is intact treatment is not required, for there is virtually no disability.

If a flexor profundus tendon alone is divided, the flexor sublimis being intact, the loss of active flexion at the distal interphalangeal joint

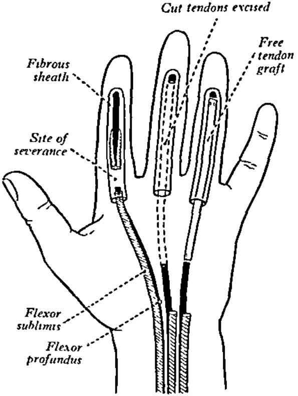

FIG. 187

Tendon graft for reconstruction of severed flexor tendons in the digital sheath. Successive stages of the operation are shown in the index, middle, and ring fingers. The use of a free graft eliminates the need for a tendon junction within the sheath

can often be accepted. Attempted repair of the tendon is better avoided because of its uncertain results. Arthrodesis of the distal interphalangeal joint in slight flexion reduces the disability to a negligible level.

If both tendons are divided operative reconstruction of the flexor profundus (not the sublimis) is advised. The reconstruction should be deferred until the skin wound is healed and the joints are mobile.

The adult type. There is complaint of tenderness at the base of the affected finger and of "locking" of the finger in full flexion (Fig. 190). The locking can be overcome either by a supreme effort or by extending the finger passively with the other hand, when the flexion is released with a distinct snap. *On examination*

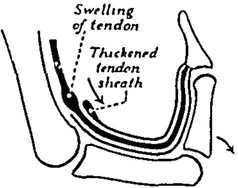

Fig. 189

Mechanism of "trigger" finger. The swollen part of the tendon is reluctant to enter the constricted mouth of the fibrous sheath. When sufficient force is exerted it enters with a snap. The thickening forms a palpable nodule at the base of the finger.

Fig. 190

"Trigger" finger. The fingers can be flexed without difficulty, but when the patient attempts to straighten them the affected finger sticks in the position shown.

there is a palpable nodule, usually slightly tender, at the base of the affected finger or thumb—that is, over the mouth of the fibrous sheath. The snapping cannot be reproduced on passive movements: it can be demonstrated only when the patient flexes the finger fully with its own muscles.

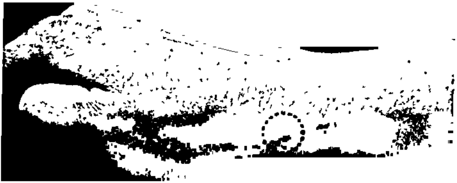

FIG. 188
Site of tenderness in De Quervain's tenovaginitis.

sheaths are usually palpable as a firm nodule. Passive adduction of the wrist or thumb causes the patient to wince with pain.

Diagnosis. The clinical picture is so characteristic that, provided the condition is borne in mind, its diagnosis presents little difficulty.

Treatment. There is a tendency to very slow natural recovery with rest But operation provides so certain a cure that it should always be advised if the disability is severe. All that is necessary is to slit or " de-roof " the offending tendon sheaths.

DIGITAL TENOVAGINITIS STENOSANS

("Trigger" finger; snapping finger)

In this rather common condition thickening and constriction of the mouth of a fibrous digital sheath interferes with the free gliding of the contained flexor tendons.

Cause. This is unknown.

Pathology. The proximal part of the fibrous flexor sheath at the base of a finger or thumb is thickened and the mouth of the sheath is constricted. The contained tendons become "waisted" opposite the constriction, and swollen proximal to it. The swollen segment enters the mouth of the sheath only with difficulty when an attempt is made to straighten the finger from the flexed position (Fig. 189).

Clinical features. The condition occurs 1) in the fingers of the middle-aged (especially women), and 2) in the thumb in infants or young children.

the major nerve trunks. A well known example is "crutch palsy," in which there is weakness or paralysis of the extensor muscles of the wrist, fingers, and thumb from repeated pressure of a crutch upon the radial nerve in the axilla. Occasionally the axillary artery has been injured in the same way, with consequent ischaemic manifestations in the digits.

DISORDERS OF THE ELBOW

Affections of the elbow may be associated with a vague referred pain in the forearm. This is especially true of "tennis" elbow, in which the pain extends along the extensor aspect of the forearm, often to the hand. Almost always, however, the local symptoms in the elbow overshadow the referred symptoms, so mistakes in diagnosis are unlikely.

In the condition of friction neuritis of the ulnar nerve, however, the symptoms and signs are predominantly in the hand, and one might suspect a local disorder of the hand itself if the possibility of a lesion at the elbow were not considered and investigated.

The infantile type (contracted thumb of infants). The infant is unable to straighten the thumb, which is locked in flexion. *On examination* it may or may not be possible to extend the thumb passively. A palpable nodule is present at the base of the thumb in the position of the mouth of the fibrous flexor sheath. It should be noted that this condition in infants is often mistaken for a dislocated thumb or a congenital deformity.

Treatment. Both the adult and the infantile type can be cured by the simple operation of incising the mouth of the fibrous flexor sheath longitudinally.

EXTRINSIC DISORDERS SIMULATING DISEASE OF THE FOREARM OR HAND

DISORDERS OF THE NECK

Certain disorders of the neck interfere with the brachial plexus or its roots, and thereby produce their predominant symptoms—or even their only symptoms—in the lower arm or hand. By far the commonest of such disorders are prolapse of a cervical intervertebral disc and osteoarthritis of the cervical spine (*cervical spondylosis*). Less common as causes of peripheral symptoms are cervical rib, tumours of the spinal column or of the spinal cord, and soft-tissue tumours involving nerves. All these conditions were described in Chapter III.

Rarely, neck disorders affect the lower arm or hand by interfering with the subclavian artery. Examples are occasionally seen in cases of cervical rib, or when the artery is obstructed by a tumour or aneurysm.

TUMOUR AT THE THORACIC INLET

A mass at the thoracic inlet is an occasional cause of peripheral symptoms in the upper limb. The commonest cause is an apical tumour of the lung (Pancost's tumour) involving the nerves of the brachial plexus.

DISORDERS OF THE UPPER ARM

Rarely, a disorder of the upper arm may produce its chief effects in the lower arm or hand, usually through the medium of

TABLE IX

ROUTINE CLINICAL EXAMINATION IN SUSPECTED DISORDERS OF THE HIP

1. LOCAL EXAMINATION OF THE HIP REGION

(Patient recumbent)

Position of pelvis

Determine the lie of the pelvis and set it square with the limbs if possible

Inspection

Bone contours and alignment
Soft-tissue contours
Colour and texture of skin
Scars or sinuses

Palpation

Skin temperature
Bone contours
Soft-tissue contours
Local tenderness

Measurements of limb length

Real or true length

Anterior superior iliac spine to medial malleolus (Angle between pelvis and limbs to be equal on each side)

If discrepancy found, determine site of shortening

(*a*) Above trochanter (Bryant's triangle, Nelaton's line, Schoemaker's line)
(*b*) Below trochanter (measure each bone)

"Apparent" or false discrepancy:

Xiphisternum to medial malleolus. (Limbs to be parallel and in line with trunk)

Examination for fixed deformity

Including Thomas's manoeuvre for detection and measurement of fixed flexion deformity

Movements (active and passive)

Flexion
Abduction; abduction in flexion
Adduction
Medial rotation; lateral rotation
Extension (with patient prone)

Power (tested against resistance of examiner)

Estimate strength of each muscle group

Examination for abnormal mobility

Test for longitudinal ("telescopic") movement

(Patient standing)

Examination for postural stability

Trendelenburg's test

Gait

2. EXAMINATION OF POTENTIAL EXTRINSIC SOURCES OF HIP SYMPTOMS

This is important if no satisfactory explanation for the symptoms is found on local examination. The investigation should include 1) the spine and sacro-iliac joints, and 2) the abdomen and pelvis.

3. GENERAL EXAMINATION

General survey of other parts of the body. The local symptoms may be only one manifestation of a widespread disease.

CHAPTER EIGHT

The Hip Region

THE hip presents some of the most fascinating problems in the whole field of orthopaedic surgery. Practically and economically, its injuries and diseases are important because they so often cause prolonged suffering and serious disablement. Academically, the region is of interest for several reasons: the mechanics of the joint are complex; it is one of the most difficult joints to examine with accuracy; and—of special significance to students—cases of hip disease are often presented as tests of clinical acumen in the examinations in surgery.

SPECIAL POINTS IN THE INVESTIGATION OF HIP COMPLAINTS

History

The characteristics of hip pain. Pain in the region of the hip is notoriously misleading, for often it is referred from the spine or pelvis and has no connection with the hip itself. Therefore one must always be

TABLE VIII

USUAL AGE INCIDENCE OF COMMON HIP DISORDERS

Age at Onset of Symptoms (Years)	Disease
0 to 2	Congenital dislocation
2 to 5	Tuberculous arthritis
5 to 10	Perthes' disease
10 to 20	Slipped upper femoral epiphysis
20 to 50	Osteoarthritis (secondary to previous injury or disease)
50 to 100	Osteoarthritis (primary)

cautious in attributing such pain to a hip lesion without first investigating the possibility of an extrinsic cause.

Pain arising in the hip is felt mainly in the groin and in the front of

understood. Accuracy in measurement is of more than academic significance; it is of practical importance when corrective operations or adjustments to the shoes are contemplated.

It is necessary to measure, first, the real or *true length* of each limb. Secondly, it is necessary to determine whether there is any "*apparent*" or *false discrepancy* in the length of the limbs from fixed pelvic tilt

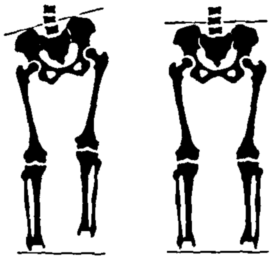

FIG. 192 FIG. 193

Figure 192—To show how pelvic tilt causes "apparent" discrepancy in limb length. Figure 193—If the pelvis is square with the limbs there can be no "apparent" discrepancy in limb length

(Figs 192-193). Whereas it is always necessary to measure the true length, it is necessary to measure "apparent" discrepancy only when there is an incorrectible pelvic tilt.

Measurement of true length. Ideally it would be desirable to measure from the normal axis of hip movement—that is, the centre of the acetabulum—but since there is no surface landmark at that point it is impracticable to do so. The measurement is therefore taken from the nearest convenient landmark—namely, the anterior superior spine of the ilium Distally, measurement is usually made to the medial malleolus.

The anterior superior spine, be it noted, is well lateral to the axis of hip movement This makes no matter if the angle between limb and pelvis is the same on each side But it will render the measurements fallacious if the angle between limb and pelvis is not the same on each side. This will be understood best by reference to Figure 194. It will be seen that abduction of a limb brings the medial malleolus

inner side of the thigh. Pain is often referred also to the knee; indeed pain in the knee is sometimes the predominant feature. In contrast, the "hip" pain that is referred from the spine is felt mainly in the gluteal region, whence it often radiates down the back or outer side of the thigh.

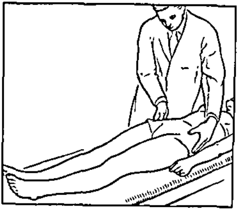

FIG. 191

First step in the clinical examination of the hip: determining the lie of the pelvis.

True hip pain is made worse by walking, whereas gluteal pain referred from the spine is aggravated by activities such as stooping and lifting, and it is often eased by walking.

Age incidence of hip disorders. Many of the important disorders of the hip occur in childhood, and often at a particular period of childhood. So true is this with some disorders that the age of the patient at the onset of symptoms affords some indication of the likely nature of the trouble, as shown in Table VIII.

Exposure

For the proper examination of the hip the patient should be stripped except for a pelvic slip or underpants, and, in women, a brassiere. The first part of the examination is conducted with the patient lying; afterwards he is examined standing and walking.

Steps in Clinical Examination

A suggested routine for clinical examination of the hip is summarised in Table IX.

Setting the Pelvis Square

This is an important preliminary step. Determine from the position of the anterior superior iliac spines whether or not the pelvis is lying square with the limbs (Fig. 191). If it is not, an attempt is made to set it square. If this is impossible it means that there is incorrectible adduction or abduction at one or other hip: in that event the fact that the pelvis is tilted should be noted and borne in mind during the subsequent steps of the examination.

Measuring the Length of the Limbs

The methods of measurement of the lower limbs are often confusing to the uninitiated, but it is important that they should be properly

understood. Accuracy in measurement is of more than academic significance; it is of practical importance when corrective operations or adjustments to the shoes are contemplated.

It is necessary to measure, first, the real or *true length* of each limb. Secondly, it is necessary to determine whether there is any "*apparent*" or *false discrepancy* in the length of the limbs from fixed pelvic tilt

FIG. 192

FIG. 193

Figure 192—To show how pelvic tilt causes "apparent" discrepancy in limb length Figure 193—If the pelvis is square with the limbs there can be no "apparent" discrepancy in limb length.

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The anterior superior spine, be it noted, is well lateral to the axis of hip movement. This makes no matter if the angle between limb and pelvis is the same on each side. But it will render the measurements fallacious if the angle between limb and pelvis is not the same on each side. This will be understood best by reference to Figure 194. It will be seen that abduction of a limb brings the medial malleolus

nearer to the corresponding anterior superior spine, whereas adduction of the limb carries the medial malleolus away from the anterior superior spine. Thus if measurements are made while the patient lies with one hip adducted and the other abducted (a common posture in cases of hip disease) inaccurate readings will be obtained: the length will be exaggerated on the adducted side and belittled on the abducted side.

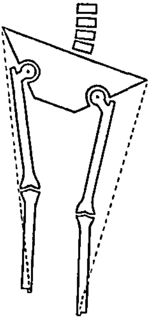

Fig. 194

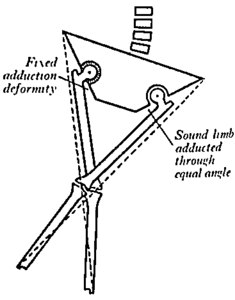

Fig. 195

Figure 194—Since the anterior superior spine is lateral to the hip joint abduction approximates the foot to it and adduction carries the foot away from it. For this reason measurements of true length are inaccurate if the angle of abduction or adduction is not equal on the two sides. Figure 195—Correct way of measuring true length when there is a fixed adduction deformity of one hip. The other hip must be adducted through an equal range. (Position of tape-measure shown by interrupted lines).

The rule is, therefore, that *to obtain an accurate comparison of their true length the two limbs must be placed in comparable positions relative to the pelvis.* Thus if one limb is adducted and cannot be brought out to the neutral position the other limb must be adducted through a corresponding angle by crossing it over the first limb before the measurements are taken (Fig. 195). Similarly, if one hip is in fixed abduction the other must be abducted through the same range before the measurements of true length are made.

Fixing the tape-measure at the anterior superior spine. A flat metal end (as found on the ordinary tailor's measure) is essential. The metal end

is placed immediately *distal* to the anterior superior spine and is pushed up against it. The thumb is then pressed firmly backwards against the bone and the tape-end together (Fig. 196). This gives rigid fixation of the tape-measure against the bone.

Taking the reading at the medial malleolus. The tip of the index finger is placed immediately distal to the medial malleolus and pushed up

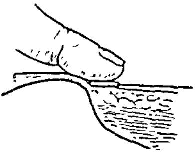

Fig 196
Fixing the tape-measure at the anterior superior spine

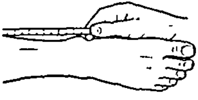

Fig. 197
Taking the measurement at the medial malleolus.

against it. The thumb nail is brought down against the tip of the index finger so that the tape-measure is pinched between them (Fig. 197). The point of measurement is indicated by the thumb nail.

Determining the site of true shortening. If measurements reveal real shortening of a limb it is necessary to determine whether the shortening is above the trochanteric level (suggesting an affection in or near the hip), or below the trochanteric level (suggesting an affection of the limb bones).

SHORTENING ABOVE THE GREATER TROCHANTER. Tests for shortening above the trochanteric level are: 1) the measurement of Bryant's triangle; 2) the construction of Nelaton's line; and 3) the construction of Schoemaker's line.

Bryant's triangle. In principle, this is nothing more than a method of comparing the distance between the greater trochanter and the wing of the ilium on the two sides [1] With the patient lying supine, a perpendicular is dropped from the anterior superior spine of the ilium towards the couch A second line is projected upwards from the tip of the greater trochanter to meet the first line at a right angle (Fig. 198). This is the important line of the triangle it is measured and compared on the two sides (The third side of the triangle is unimportant. It joins the anterior superior spine to the tip of the greater trochanter.)

[1] A simpler and equally informative method is to measure, on each side, the distance between the tip of the greater trochanter and the highest point of the iliac crest

Measurement of Bryant's triangle gives a comparison between the pelvis-to-trochanter distance on each side. Relative shortening on one side indicates that the femur is displaced upwards in consequence of a

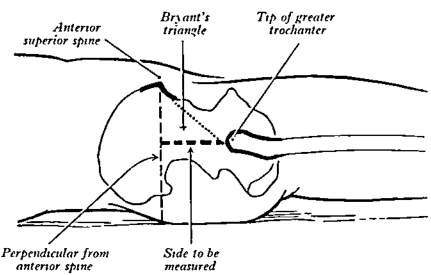

FIG 198

Bryant's triangle, which indicates whether the greater trochanter is higher on one side than the other.

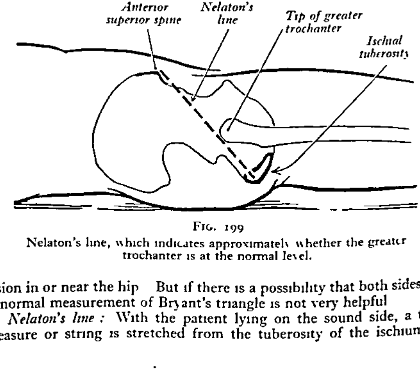

FIG. 199

Nelaton's line, which indicates approximately whether the greater trochanter is at the normal level.

lesion in or near the hip But if there is a possibility that both sides are abnormal measurement of Bryant's triangle is not very helpful

Nelaton's line: With the patient lying on the sound side, a tape measure or string is stretched from the tuberosity of the ischium to

the anterior superior spine of the ilium (Fig. 199). Normally the greater trochanter lies on or below that line. If the trochanter lies above the line the femur has been displaced upwards.

Schoemaker's line : This is a similar test. A line is projected on each side of the body from the greater trochanter through and beyond

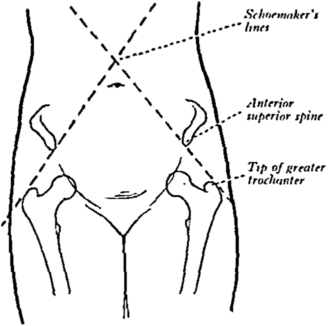

FIG. 200

Schoemaker's line in a normal subject
When the femur is displaced upwards the line passes below the umbilicus

the anterior superior spine. Normally the two lines meet in the midline above the umbilicus (Fig. 200) If one femur is displaced upwards owing to shortening above the greater trochanter the lines will meet away from the midline on the opposite side. If both femora are displaced upwards the lines will meet at or near the midline but below the umbilicus.

SHORTENING DISTAL TO THE TROCHANTER. True shortening is sometime accounted for by an abnormality below the trochanteric level, such as a congenital defect of development, impaired epiphysial growth, or a previous fracture with overlapping of the fragments. To investigate this possibility individual measurements should be made of the femur (tip of greater trochanter to line of knee joint) and of the tibia (line of knee joint to medial malleolus) on each side.

Measurement of "apparent" discrepancy in limb length. "Apparent" or false discrepancy in limb length is due entirely to

incorrectible sideways tilting of the pelvis (Fig. 192). The usual cause is a fixed adduction deformity at one hip, giving an appearance of shortening on that side, or a fixed abduction deformity, giving an

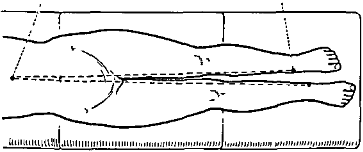

FIG. 201
For correct measurement of "apparent" discrepancy in limb length the limbs must be parallel and in line with the trunk.

appearance of lengthening. Exceptionally, fixed pelvic obliquity is caused by severe lumbar scoliosis.

To measure apparent discrepancy the limbs must be placed parallel to one another and in line with the trunk. Measurement is made from any fixed point in the midline of the trunk (for example, the xiphisternum) to each medial malleolus (Fig. 201).

If there is a discrepancy of true length it must be allowed for when "apparent" discrepancy is determined.

Examination for Fixed Deformity

Contracture of the joint capsule or of muscles may cause fixed deformity at the hip, preventing its being placed in a neutral position. Fixed flexion, fixed adduction, and fixed lateral rotation are common in some forms of arthritis.

Fixed adduction deformity. This is detected by judging the relationship between pelvis and limbs. It will already have been noted at an earlier stage of the examination. If fixed adduction is present the transverse axis of the pelvis (as indicated by a line joining the two anterior superior spines) cannot be set at right angles to the affected limb, but lies at an acute angle with it.

Fixed abduction deformity. The angle between the transverse axis of the pelvis and the limb is greater than 90 degrees.

Fixed flexion deformity. This is determined by a manœuvre known as Thomas's test. *Principle of Thomas's test:* If a patient has a fixed flexion deformity at the hip he compensates for it, when lying on his back, by arching the spine and pelvis into exaggerated lordosis (Fig. 202). This allows the affected limb to lie flat on the couch. To measure the

angle of fixed flexion deformity it is necessary to correct the lumbo-pelvic lordosis. This is done by flexing the pelvis (and with it the lumbar spine) by means of the fully flexed sound limb (Fig. 203).

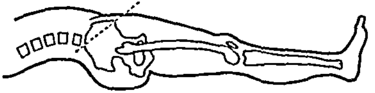

FIG. 202

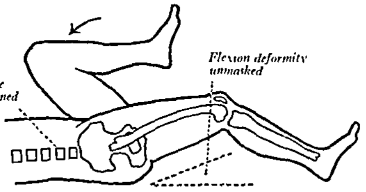

FIG. 203

Thomas's test for fixed flexion deformity Figure 202—Flexion deformity masked by arching of spine and pelvis. Figure 203—Deformity revealed by flexing the sound hip and, by continuing the flexion force, correcting the arching of spine and pelvis

Technique of the manoeuvre · One hand is placed behind the lumbar spine (between it and the couch) to assess the degree of lumbar lordosis. If there is no lordosis when the affected limb lies flat on the couch there can be no fixed flexion deformity and there is no need to proceed with the test If there is excessive lordosis, as indicated by arching of the back, it is corrected in the following way · The *sound* hip is flexed to the limit of its range The limb is then pushed further into flexion, thereby rotating the pelvis on a horizontal transverse axis until the arching of the spine is obliterated. During this manoeuvre the disordered limb, if in fixed flexion, is automatically lifted from the couch as the lumb[illegible]
the limb is r
Fixed lateral

of the thigh is the patella, which normally points forwards when the hip is in the neutral position. If there is fixed lateral rotation the limb cannot be rotated to the neutral position. The angle by which it falls short of the neutral when rotated medially as far as possible is the angle of fixed lateral rotation deformity.

Movements

The accurate determination of hip movements demands much care, for a restriction of true hip movement is easily masked by movement of the pelvis. It is therefore essential to place one hand upon the pelvis to detect any movement there, while the other guides and supports the limb. *Flexion*. Movement of the pelvis is best detected by grasping the crest of the ilium (Fig. 204). Only in this way is it possible to differentiate between true hip movement and the false flexion imparted by rotation of the pelvis. The normal range of true hip flexion is about 120 degrees, but it varies according to the build of the patient.

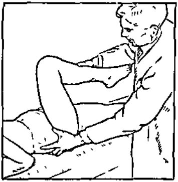

FIG. 204

Testing hip flexion. The right hand supports the limb while the left grips the ilium to detect pelvic rotation

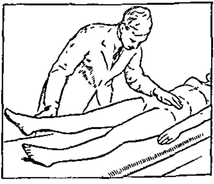

FIG. 205

Testing abduction of the hip. The right hand supports the limb while the left, bridging the two anterior superior spines, is ready to detect tilting of the pelvis

Abduction. The limb to be tested is supported by one hand while ... spine to anterior ... t the hip can be ... by tilting of the pelvis. The normal range of true abduction at the hip is 30 to 40 degrees (more in children). *Abduction in flexion*. This is often the first movement to suffer restriction in arthritis of the hip. The patient flexes his hips and knees by drawing the heels towards the buttocks. He then allows the knees to fall away from one another towards the couch. The normal range is about 70 degrees (90 degrees in young children). *Adduction*. The limb to be examined is crossed over the other limb. Again care must be taken to differentiate between true

adduction and the false movement imparted by tilting of the pelvis. The normal range of adduction is about 30 degrees. *Lateral rotation and medial rotation :* Judge the range by an imaginary pointer thrust axially into the patella, not by the position of the foot. The normal range both of medial and of lateral rotation is about 40 degrees. *Extension :* This is estimated with the patient lying prone. Normally the hip extends about 5 degrees beyond the neutral.

Examination for Abnormal Mobility

In cases of marked instability of the hip longitudinal movement or "telescoping" can sometimes be demonstrated—especially in children with congenital dislocation of the hip. To carry out the test the limb is grasped firmly in one hand and alternately pushed and pulled in its long axis, the trunk being steadied by the examiner's other hand upon the iliac crest.

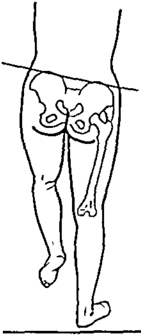

FIG. 206

Negative Trendelenburg test. The hip abductors are acting normally, tilting the pelvis upwards when the opposite leg is raised from the ground.

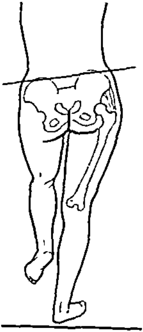

FIG. 207

Positive Trendelenburg test. The hip abductors are unable to control the dropping of the pelvis when the opposite leg is raised.

Examination for Postural Stability; the Trendelenburg Test

The Trendelenburg manoeuvre is a test of the stability of the hip, and particularly of the ability of the hip abductors (gluteus medius and

gluteus minimus) to steady the pelvis upon the femur. *Principle of the test :* Normally, when one leg is raised from the ground the pelvis tilts upwards on that side, through the action of the hip abductors of the standing limb (Fig. 206). (This automatic mechanism allows the lifted leg to clear the ground in walking.) If the abductors are inefficient they are unable to sustain the pelvis against the body weight and it tilts downwards instead of rising on the side of the lifted leg (Fig. 207) *Technique :* Stand behind the patient. Instruct him first to stand upon the sound limb and to raise the other from the ground. Having thus got the idea of what he is required to do, he should now stand on the affected limb and lift the sound leg from the ground. By inspection, or by palpation with a hand upon the iliac crest, observe whether the pelvis rises or falls on the lifted side. Remember that the limb upon which the patient stands is the one under test. If the pelvis rises on the opposite side (normal) the test is negative (Fig. 206) *If it falls, the test is positive (Fig. 207) ; in other words the abductor muscles* are incapable of stabilising the pelvis upon the femur. *Causes of positive Trendelenburg test :* There are three fundamental causes : 1) paralysis of the abductor muscles (example—poliomyelitis) ; 2) marked approximation of the insertion of the muscles to their origin by upward displacement of the greater trochanter, so that the muscles are slack (examples—severe coxa vara ; congenital dislocation of the hip) ; 3) absence of a stable fulcrum (example—ununited fracture of the femoral neck).

Gait

Watch how the patient stands and observe his gait on walking. Note that a patient with an unstable or painful hip prefers to use a stick in the *opposite* hand.

Extrinsic Causes of Pain in the Hip Region

If examination of the hip itself fails to reveal abnormalities sufficient to account for the patient's symptoms, possible causes outside the hip must be investigated. Attention should be directed particularly to the spine and sacro-iliac joints (including a neurological survey of the

Radiographic Examination

Routine radiographs should include an antero-posterior projection showing the whole pelvis with both hips, and lateral projections of each hip.

In special cases there is a place for stereoscopic films and for arthrography (that is, radiography after the intra-articular injection of a radio-opaque fluid) When there is a possibility that the symptoms may be referred from the back additional radiographs of the spine and sacro-iliac joints must be obtained.

CLASSIFICATION OF DISORDERS IN THE HIP REGION

ARTICULAR DISORDERS OF THE HIP

CONGENITAL DEFORMITIES

Congenital dislocation of the hip

ARTHRITIS

Transient arthritis of children
Pyogenic arthritis
Rheumatoid arthritis
Tuberculous arthritis
Osteoarthritis
Charcot's osteoarthropathy

OSTEOCHONDRITIS

Perthes' disease

MECHANICAL DISORDERS

Slipped upper femoral epiphysis

EXTRA-ARTICULAR DISORDERS IN THE REGION OF THE HIP

DEFORMITIES

Coxa vara

INFECTIONS

Osteomyelitis of the upper femoral metaphysis
Extra-articular tuberculosis of bone
Tuberculosis of the trochanteric bursa

MECHANICAL DISORDERS

Snapping hip

ARTICULAR DISORDERS OF THE HIP

CONGENITAL DISLOCATION OF THE HIP

This is a spontaneous dislocation of the hip occurring either before birth or shortly afterwards, in consequence of an underlying dysplasia of the joint.

Cause. The cause is unknown. Hereditary factors play a part.

Pathology. *The femoral head :* The ossific nucleus is late in appearing and its development is retarded. The femoral head is dislocated upwards and laterally from the acetabulum. *The femoral neck :* In most cases the neck is anteverted beyond the normal angle for infants of 25 degrees. *The acetabulum :* The ossific centre for the roof of the acetabulum, like that for the femoral head, is under-developed. The bone slopes upwards at a steep angle instead of forming a nearly horizontal roof for the acetabulum. T

formed at first, b

ment does not p

contour with ste

elongated as the

cartilaginous limbus : This is commonly displaced from the rim of the acetabulum into the socket. In some cases it may possibly form an obstacle to reduction of the dislocation.

Clinical features. Girls are affected five times as often as boys. In one-third of all cases both hips are affected. Usually no abnormality is noticed until the child begins to walk. Walking is often delayed, and there is a limp or a waddling gait. *On examination*, the main features in unilateral cases are asymmetry (especially of the buttock folds) and shortening of the affected limb (Fig. 208) In bilateral cases the main feature is widening of the perineum. In most cases the affected limb is abnormally mobile in its long axis (" telescopic " movement). The femoral head is often palpable in an abnormal position. *Radiographic examination :* There are three important radiographic features (Figs. 209-210) (1) The ossific centre for the head of the femur is late in appearing and its development is retarded ; (2) the bony acetabular roof has a pronounced upward slope ; and (3) the femoral head (as judged by the position of the ossific centre) is displaced upwards and laterally from its normal position in the centre of the acetabulum. *Arthrography* (radiography after the injection of opaque fluid into the joint) is useful in showing the outline of the cartilaginous elements.

Diagnosis. Asymmetry of the hips should be looked for as a routine in newborn infants. Delay in beginning to walk or abnormality of gait in early childhood should invariably arouse suspicion of congenital dislocation of the hip, and radiographic

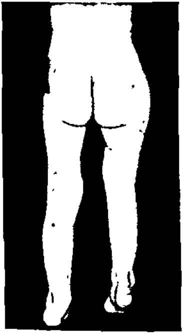

FIG. 208

FIG. 209

Figure 208—Congenital dislocation of the right hip. Note the shortening of the limb and the asymmetry of the buttock and thigh creases Figure 209—Radiograph at age 1½ years.

FIG. 210

Congenital dislocation of the . . . .

Pathology. (1) *The femoral head :* The ossific nucleus is late in appearing and its development is retarded. The femoral head is dislocated upwards and laterally from the acetabulum. *The* (2) *femoral neck :* In most cases the neck is anteverted beyond the normal angle for infants of 25 degrees. (3) *The acetabulum :* The ossific centre for the roof of the acetabulum, like that for the femoral head, is under-developed. The bone slopes upwards at a steep angle instead of forming a nearly horizontal roof for the

[illegible]

cartilaginous limbus : This is commonly displaced from the rim of the acetabulum into the socket. In some cases it may possibly form an obstacle to reduction of the dislocation.

Clinical features. Girls are affected five times as often as boys. In one-third of all cases both hips are affected. Usually no abnormality is noticed until the child begins to walk. Walking is often delayed, and there is a limp or a waddling gait. *On examination*, the main features in unilateral cases are asymmetry (especially of the buttock folds) and shortening of the affected limb (Fig. 208). In bilateral cases the main feature is widening of the perineum. In most cases the affected limb is abnormally mobile in its long axis ("telescopic" movement). The femoral head is often palpable in an abnormal position. *Radiographic examination :* There are three important radiographic features (Figs. 209-210) (1) The ossific centre for the head of the femur is late in appearing and its development is retarded ; (2) the bony acetabular roof has a pronounced upward slope ; and (3) the femoral head (as judged by the position of the ossific centre) is displaced upwards and laterally from its normal position in the centre of the acetabulum. *Arthrography* (radiography after the injection of opaque fluid into the joint) is useful in showing the outline of [illegible]

Diagnosis [illegible] hould be looked for as a routine in [illegible] in beginning to walk or (5) abnormality of gait in early childhood should invariably arouse suspicion of congenital dislocation of the hip, and radiographic

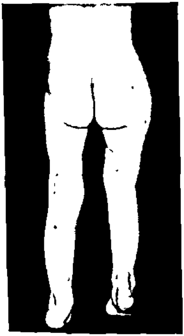

FIG 208 FIG. 209

Figure 208—Congenital dislocation of the right hip. Note the shortening of the limb and the asymmetry of the buttock and thigh creases. Figure 209—Radiograph at age 1½ years

FIG. 210

examination should be insisted upon. It is far better to waste a hundred or even a thousand x-ray films than to overlook one congenitally dislocated hip. Indeed a case could be made for routine radiography, on miniature films, of every child between the ages of six months and a year.

Course and prognosis. The earlier the dislocation is reduced the better the prognosis. But at best only about half the treated patients remain free from trouble throughout life. Gradual redislocation is all too frequent, and pain from secondary degenerative changes often develops in middle adult life.

Treatment. This varies according to the age of the patient when advice is sought.

1) *Neonatal cases* (within six months of birth). The dislocation is reduced by abduction of the hip. Abduction is maintained by a V-shaped splint or a plaster for six months.

2) *Age 6 months to 4 years.* Conservative treatment is adopted unless indications for operation arise. The dislocation is reduced by gentle manipulation, preceded by weight traction on an abduction frame for four weeks. Reduction is maintained by immobilising the hips in a plaster which holds both limbs abducted 80 degrees. The plaster is retained for one to one and a half years, to allow the acetabulum to develop adequate depth. If after removal of the plaster the femoral head begins gradually to redislocate, operation is undertaken to stabilise the hip by levering down a rim of bone and cartilage from the ilium to widen the acetabular roof and make it) (Fig. 211). If there l neck it is corrected by reatment is complete.

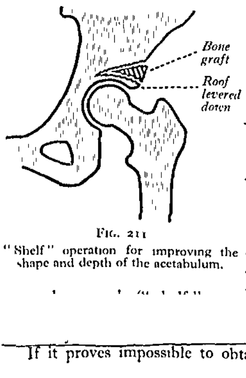

FIG. 211

"Shelf" operation for improving the shape and depth of the acetabulum.

If it proves impossible to obtain a satisfactory reduction by

traction and manipulation, operative reduction should be proceeded with, as for the next group.

Age 4 to 8 years. Operative reduction is undertaken, because the dislocation cannot be fully reduced by manipulation. At the same time a "shelf" is levered down to widen the roof of the acetabulum. The limb is rested in plaster for three months, after which a rotation osteotomy is done if necessary to correct excessive anteversion of the femoral neck. Walking is encouraged as soon as the osteotomy is united.

Age 8 to 16 years. In patients of this age normal function cannot be restored to the hip even though the dislocation be reduced. But unless something is done the femur will gradually migrate high up on the ilium, with permanent establishment of the rather grotesque deformity characteristic of old unreduced dislocation of the hip. Therefore operation is advisable to prevent this progressive displacement and to make the conditions favourable for reconstructive surgery in adult life. The acetabulum is deepened by gouging out its cartilaginous floor, and the dislocation is reduced after interposing a layer of capsule between the femoral head and the acetabulum, to serve as a lining membrane. This technique—a form of arthroplasty—is known as the Colonna operation.

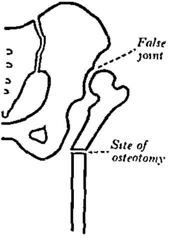

FIG 212
Abduction osteotomy (Schanz) for painful unreduced congenital dislocation in adults.

Age 16 years onwards After adolescence no treatment is advised unless secondary degenerative changes lead to severe pain. If increasing pain justifies operative treatment, the choice of method depends largely upon whether the dislocation affects one or both hips. If only one hip is affected arthrodesis is recommended. If both hips are affected abduction osteotomy at the level of the ischial tuberosity (Schanz) should usually be advised (Fig. 212) This operation helps to stabilise the hip by preventing adduction of the pelvis on the femur, thereby eliminating the

Trendelenburg " dip " on walking. At the same time the adducted upper fragment lying close to the side wall of the pelvis affords it greater support, and stability is further improved by the medial shift of the femoral shaft.

TRANSIENT ARTHRITIS OF THE HIP

(Traumatic synovitis of the hip)

The so-called transient arthritis of childhood is a short-lived affection of the hip of uncertain pathology, characterised clinically by pain, limp, and limitation of hip movements.

Cause. This is unknown. Injury is possibly a factor but the evidence in support of it is slender.

Pathology. This is unknown. It is assumed that there is a mild inflammation of the synovial membrane, possibly initiated by injury.

Clinical features. The condition is virtually confined to children under 10, especially boys. The child complains of pain in the groin and thigh and he is noticed to limp. *On examination* the only physical sign is limitation of hip movements. *Radiographs* show no alteration from the normal.

Diagnosis. The condition is important only because it resembles clinically the early stages of tuberculous arthritis of the hip and Perthes' disease Transient arthritis should be diagnosed only after the hip has recovered—never while the symptoms and signs are present. While the symptoms and signs last the case should be regarded as one of suspected tuberculous arthritis and the child placed under observation in bed ; full recovery within four weeks excludes tuberculous arthritis and justifies a retrospective diagnosis of transient arthritis

Course. Full recovery, with return of a normal range of hip movements, invariably occurs within four weeks.

Treatment. A few weeks' rest in bed is the only treatment required.

PYOGENIC ARTHRITIS OF THE HIP

(General description of pyogenic arthritis, p. 31.)

Pyogenic arthritis of the hip is uncommon. It occurs mainly in children, in whom it is often secondary to osteomyelitis of the upper end of the femur. The disease has special characteristics in young infants.

Pathology. The organisms (usually staphylococci or streptococci) may reach the joint directly through the blood stream, or the infection may spread from an adjacent focus of osteomyelitis. Rarely a penetrating wound is responsible. There is an acute inflammatory reaction in the joint tissues, with an effusion of turbid fluid or pus. An abscess may track to the surface of the buttock or thigh. In favourable cases healing with restoration to normal can occur, but in many cases the joint is permanently destroyed or damaged. In infants bony ankylosis never occurs because the femoral head and the acetabulum are composed almost entirely of cartilage rather than bone, but there is often total destruction of the developing femoral head with secondary dislocation of the hip. In older children and adults there may be either bony or fibrous ankylosis.

Clinical features. The clinical features differ so much in infants and in older subjects that separate descriptions are required.

PYOGENIC ARTHRITIS OF INFANTS. The onset is within the first year of life. Often there has been a known septic lesion somewhere on the body (for example, umbilical sepsis) but it may have caused little anxiety. Then the child becomes unwell and pyrexial. *On examination* it is not always apparent at first that the hip is the seat of the trouble. But careful examination will show thickening in the hip area, and movements of the joint are restricted. Sometimes an abscess points at the skin surface in the buttock or thigh. *Radiographic examination :* No change is seen in the early stages. If the infection progresses to the stage of destroying the capital epiphysis of the femur the ossific nucleus fails to appear as it should at the age of one year. In such a case subsequent radiographs will show gradual dislocation of the hip. This "pathological" dislocation is distinguished from congenital dislocation by the facts that the acetabular roof is of normal shape and that the capital epiphysis is permanently absent

FIG 213

Pyogenic arthritis of the hip in an infant The epiphysis of the head of the femur has been destroyed and the hip is dislocated. Note the normal appearance of the acetabular roof, which distinguishes this from a congenital dislocation

(Fig 213). *Investigations* · Aspiration of the hip yields pus from which the causative organism can be identified

PYOGENIC ARTHRITIS IN OLDER CHILDREN AND ADULTS. The onset is acute or subacute, with pain in the hip made worse by attempted weight-bearing, and severe limp. There is constitutional disturbance with pyrexia *On examination* there is a fullness around the hip region from swelling of the joint. All movements of the hip are markedly restricted, and painful if forced *Radiographic examination* · There is no change in the early stages. Later, if the infection persists,

FIG 214

FIG 214

Bony ankylosis of the hip caused by pyogenic arthritis. The infection spread to the hip from a focus of osteomyelitis in the upper metaphysis of the femur.

there are osteoporosis and narrowing of the cartilage space. Finally, there may be bony ankylosis of the joint (Fig. 214). *Investigations:* The erythrocyte sedimentation rate is raised. There is a polymorphonuclear leucocytosis. Aspiration of the joint yields pus from which the causative organism can be identified.

Treatment. *Constitutional treatment* is by rest and systemic chemotherapy with penicillin or other appropriate antibiotic. *Local treatment:* Rest for the joint and relief of muscle spasm are best ensured by weight traction through adhesive skin strapping applied to the leg. The joint is aspirated daily until pus ceases to re-form; after each aspiration a solution of penicillin or other appropriate antibiotic is injected into the joint. In very severe cases more effective drainage can be obtained by wide incisions into the joint. When the infection has been overcome active movements are encouraged.

Pathological dislocation complicating pyogenic arthritis of infants. Final treatment of this crippling condition must await adolescence. In childhood the aim of treatment is to prevent progressive upward displacement of the femur and thereby to minimise the shortening. Provided the infection has settled, operation should be undertaken to deepen the acetabulum, to place the upper end of the femur within it, and, if necessary, to lever down a " shelf " of ilium over it. In adolescence, when the bones are nearing full development, arthrodesis of the hip is recommended

RHEUMATOID ARTHRITIS OF THE HIP

(General description of rheumatoid arthritis, p. 34.)

The hip joints often escape in cases of rheumatoid polyarthritis. On the other hand when they are affected the consequent disability is serious

Pathology. This is the same as that of rheumatoid arthritis in other joints. In long-established cases degenerative changes are superimposed upon the original inflammatory condition, giving rise to secondary osteoarthritis

Clinical features. The changes may affect one or both hips, in common with several other joints. The main symptoms are pain and limitation of movement, aggravated by activity. *On*

examination swelling is not obvious because the joint is so dee situated ; for the same reason the temperature of the overly skin is not increased as it is in rheumatoid affection of the m superficial joints. The range of all hip movements is impai and movement is painful if forced. A fixed flexion deformity adduction deformity may develop The gluteal and thigh musc

FIG 215

Long-established rheumatoid arthritis of the hips, with destruction of cartilage space The marginal osteophytes indicate that osteoarthritis becoming superimposed upon the old rheumatoid disease

are wasted. *Radiographic examination :* At first there are radiographic changes. Later, there is diffuse osteoporosis in area of the joint. Later still, destruction of articular cartila leads to narrowing of the cartilage space between femur a acetabulum (Fig 215) *Investigations :* The erythrocyte se mentation rate is increased during the active phase.

Course. The disease becomes inactive after months or yea but the hip is seldom restored to normal Secondary degenerati changes commonly lead to the eventual development of oste arthritis, with increasing pain and disability

Treatment. Constitutional treatment is the same as that f rheumatoid arthritis in general. Local treatment for the h joints depends upon the activity and severity of the inflammato

reaction. In the worst cases rest in bed is required. But when the reaction is moderate or mild exercises and active use within the limits of pain are encouraged. Various physiotherapeutic methods can be given a trial in attempts to reduce discomfort and hasten resolution. For a deep joint such as the hip short-wave diathermy is the most effective of these measures.

Operative treatment : When pain is severe and walking is limited to a few yards operation is justified. Since many other joints are usually affected as well as the hips, conditions are seldom suitable for arthrodesis of the hip. Arthroplasty, by the insertion of a metal cup (cup arthroplasty), by replacing the whole femoral head by a metal or plastic prosthesis (replacement arthroplasty), or by excision of the head and neck of the femur, offers reasonable hope of a painless joint with a moderate range of movement (Figs. 220-222).

TUBERCULOUS ARTHRITIS OF THE HIP

(General description of tuberculous arthritis, p. 37.)

The hip is one of the joints most commonly affected by tuberculosis.

Pathology. Tubercle bacilli reach the hip through the blood stream from a focus elsewhere The synovial membrane is much thickened by the tuberculous inflammatory reaction. Unless the disease is quickly arrested the articular cartilage is destroyed and the underlying bone is eroded Abscess formation is common

Clinical features. The patient is usually a child of 2 to 5 years, sometimes an older child or young adult. There is often a history of contact with a person with active pulmonary tuberculosis. The symptoms are pain and limp The general health is usually impaired *On examination* a thickening is often palpable in the region of the hip All movements of the hip are limited, often markedly, and attempts to force movement provoke pain and muscle spasm The gluteal and thigh muscles are wasted. A " cold " abscess is sometimes palpable in the upper thigh or buttock A tuberculous lesion may be apparent elsewhere in the body. *Radiographic examination :* The earliest sign is diffuse osteoporosis in the area of the hip This is detected most easily when the bones are compared with those of the sound side, taken on the same film

FIG. 216
Early stage of tuberculous arthritis of the right hip The bone is rare but the cartilage space is not reduced The joint might be saved

FIG. 217
Tuberculous arthritis in a more advanced stage The cartilage has been destroyed and the articular surfaces of the bones have lost their sharp definition The joint is permanently destroyed

;. 216). At first the changes are slight, but it must be emphasised t even slight osteoporosis is significant. In the more fully eloped stage the osteoporosis is obvious; and later there is ızziness" *of the joint margins and narrowing of the cartilage* ce, indicating erosion of the articular cartilage (Fig. 217). :er still, bone may also be eroded; but in a favourable case the :ase is arrested before these later changes occur. *Investigations:* e erythrocyte sedimentation rate is increased. The Mantoux t is positive. Biopsy of the synovial membrane reveals the ical histological features of tuberculosis.

agnosis. This is mainly from transient arthritis, low-grade ɔgenic arthritis, and Perthes' disease (osteochondritis). In : early stages, before radiographic changes are evident, dis- ction from transient arthritis is not always possible until progress s been observed for three or four weeks. Important features pporting a diagnosis of tuberculosis are: a history of contact th tuberculosis (often parental); the presence of a tuberculous ion elsewhere; strongly positive Mantoux reaction (in children); " cold " abscess; the characteristic radiographic changes; a gh sedimentation rate, and the typical histological appearances biopsy of the synovial membrane.

ɔurse and prognosis. In a reasonable proportion of cases, pecially in children, the lesion is aborted by treatment and a und joint is preserved, provided there has been no destruction cartilage or bone when treatment is begun. If cartilage and ɪne are eroded the joint is permanently damaged and often totally :stroyed

reatment. In its essentials treatment is the same as that for her tuberculous joints. *Constitutional treatment* is by rest in a ɔuntry hospital, and chemotherapy with streptomycin, para- nino-salicylic acid (PAS), and isonicotinic acid hydrazide NAH). The drugs are continued for six months unless signs toxicity appear. *Local treatment* is initially by rest for the hip a frame or in plaster for a first period of six months. The ıbsequent treatment depends upon the progress made. If ıdiographs after six months show no destruction of cartilage or one, if the general health is good, and if the erythrocyte sedi- ıentation rate has shown steady improvement, the hip is left ree for a clinical trial. Joint movements are practised in bed

for a further month, and if there is no evidence of deterioration full activity is gradually resumed.

On the other hand, if at the end of the first six months' treatment radiographs show destruction of cartilage or bone (Fig. 217) all hope of preserving a movable joint is abandoned and a sound bony fusion becomes the ultimate objective. To that end immobilisation (not necessarily in recumbency) is continued until the lesion becomes quiescent, as judged from the general health, the erythrocyte sedimentation rate, and improved radiographic appearances. Finally, the joint is fused by operation, using one of the extra-articular techniques (ilio-femoral or ischio-femoral arthrodesis). In children arthrodesis should be deferred until the age of 12 years.

OSTEOARTHRITIS OF THE HIP

(General description of osteoarthritis, p. 41.)

Osteoarthritis of the hip is a common cause of severe disablement in the elderly.

Cause. It is caused by wear and tear. Any injury or disease that damages the joint surfaces accelerates the wear-and-tear process and thus predisposes to the development of osteoarthritis. Common examples are fracture of the acetabulum, Perthes' osteochondritis, and slipped upper femoral epiphysis. In another group a developmental imperfection (dysplasia) or congenital subluxation or dislocation is responsible In other cases the changes are simply a consequence of age-degeneration.

Pathology. The articular cartilage is worn away, especially at the point where most weight is transmitted. The underlying bone becomes hard and "eburnated." Hypertrophy of bone at the joint margins leads to the formation of osteophytes.

Clinical features. The patient is usually elderly , but when the osteoarthritis is secondary to previous hip disease or to injury it often arises in middle life There is pain in the groin and front of the thigh ; often also in the knee The pain is made worse by walking and eased by rest. Later, there may be complaint of stiffness, which manifests itself in everyday life by inability to reach the foot to tie the shoe laces or cut the toe nails. The

symptoms tend to increase progressively month by month and year by year until they eventually cause severe painful limp and incapacity for normal activities. *On examination* all hip movements are impaired. The limitation of abduction, adduction, and rotation is marked, but a good range of flexion is often preserved. Forced movements are painful. Fixed flexion deformity and fixed adduction deformity are common. *Radiographic examination :* The changes are characteristic. There is diminution of the cartilage space, with a tendency to sclerosis of the surface bone (Fig. 218). Hypertrophic spurring of bone (osteophyte formation) is usually seen at the joint margins

FIG. 218
Osteoarthritis of the hip. The characteristic features are narrowing of the cartilage space, sclerosis of the bone surfaces, and osteophytes at the joint margins (See also Figure 24, p. 43)

Treatment. The treatment required depends upon the severity of the disability. Mild osteoarthritis is best left untreated ; in cases of moderate severity conservative treatment suffices ; in severe cases operation is often advisable.

Conservative treatment. Clearly no form of conservative treatment can possibly influence the distorted anatomy of the joint. At best such treatment is only palliative ; it may alleviate but cannot abolish the pain. Five methods will be mentioned. 1) " *Relative* " *rest :* By this is meant a modification of the patient's mode of life by change of occupation or adjustment of duties so that the work thrown upon the hip is reduced 2) *Drugs :* Mild analgesics are often helpful, especially when the pain disturbs sleep. 3) *Physiotherapy :* Local deep heat by short-wave diathermy, with exercises to strengthen the muscles and to preserve mobility, often produces temporary relief 4) *Injections into the joint :* The injection of local anaesthetic solutions or of weak acids (to counteract alleged

excessive alkalinity of the joint fluid) have been recommended. Their efficacy is extremely doubtful. 5) *Deep x-ray therapy:* This is occasionally worth a trial in severe cases when operation is considered inadvisable.

Operative treatment. This may be palliative, to alleviate the pain, or radical, to abolish it. Again five methods are available; the first three are palliative, and the last two radical. 1) *Capsulectomy:* Excision of the joint capsule relieves that proportion of the pain which is transmitted through the capsular nerve endings. In time the capsule grows again and the pain recurs. 2) *Denervation:* Severance of the obturator nerve and the nerve to the quadratus femoris cuts off a large part (but not all) of the sensory nerve supply of the hip. The relief obtained is incomplete and usually only temporary. 3) *Osteotomy* (McMurray displacement osteotomy): The femur is divided at the level of the lower margin of the acetabulum and the shaft fragment is displaced medially to lie close under the acetabulum (Fig. 219). In consequence weight is transmitted more directly through the pelvis to the femoral shaft, and the mechanical strain upon the hip joint is reduced Relief is often satisfactory and some joint movement is retained. 4) *Arthrodesis:* The joint is fused in the neutral position or in very slight flexion (Fig. 4, p. 19). There is complete relief of pain, and good function is possible so long as the other hip and the knees are normal. 5) *Arthroplasty:* New joint surfaces are created either by fitting a highly polished metal cup, which moves freely upon the femoral head and within the acetabulum (cup arthroplasty) (Fig. 220), or by removing the head and fitting a prosthesis, which remains more or less fixed to the neck of the femur (replacement arthroplasty) (Fig. 221). Relief from pain is often complete and function good;

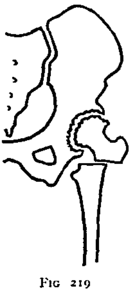

FIG 219

FIG 219

Displacement osteotomy of the femur (McMurray) The medial displacement of the distal fragment causes the line of weight-bearing to pass directly through the joint to the axis of the femoral shaft, so reducing leverage stresses.

but a satisfactory result cannot be assured in every case. A third form of arthroplasty is that known as excision arthroplasty: a false joint is created by excising the head and neck of the femur (Girdlestone pseudarthrosis) (Fig. 222). The resulting joint is usually painless and mobile, though it tends to be rather unstable.

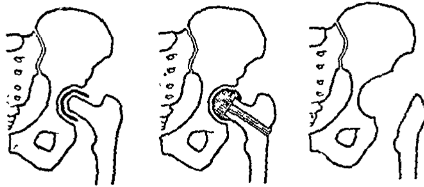

FIG. 220 FIG 221 FIG. 222

Three methods of arthroplasty of the hip. Figure 220—Cup arthroplasty. Figure 221—Replacement arthroplasty. Figure 222—Excision arthroplasty (Girdlestone pseudarthrosis)

Choice of method. When the patient is reasonably fit the more radical operation of arthrodesis or arthroplasty should always be preferred to the palliative operations. If only one hip is affected arthrodesis is the most reliable method. If both hips are affected arthrodesis may be done on one side and arthroplasty or osteotomy on the other. All the factors of the case must be weighed up carefully before a final decision is made.

CHARCOT'S OSTEOARTHROPATHY OF THE HIP

(General description of Charcot's osteoarthropathy, p. 47.)

This is a sequel to tabes dorsalis. It is much less common in the hip than in the knee. Clinically the hip becomes unstable and the limb is shorter than its fellow. Pain is slight or absent. *Radiographs* show marked destruction of the joint, which is often subluxated or dislocated. There may be massive formation of new bone round the joint. Neuropathic changes may be present also in the knee, spine, or other joints.

Treatment is palliative. a weight-relieving caliper should be fitted.

PERTHES' DISEASE

(Legg-Perthes' disease coxa plana; pseudocoxalgia; osteochondritis of the femoral capital epiphysis)

Perthes' disease is osteochondritis of the epiphysis of the femoral head. The general features of osteochondritis were described in Chapter II (p. 77). Like osteochondritis elsewhere, Perthes' disease is an affection of childhood. The femoral head is temporarily softened and may become deformed. The main importance of the condition is that it may lead to the later development of osteoarthritis of the hip.

Cause. This is unknown. A local disturbance of blood supply is believed to play a part.

Pathology. The bony nucleus of the epiphysis becomes soft and granular. While in this state it is readily squashed if subjected to pressure such as that entailed by weight-bearing. Eventually the bone hardens again, but if it has been allowed to deform it never regains its normal shape. The whole cycle of softening and re-hardening occupies about two years.

Clinical features. The disease is almost confined to children of 5 to 10 years. It usually affects only one hip. The child complains of pain in the groin or thigh and is noticed to limp. There is no disturbance of general health. *On examination* the only striking sign is moderate limitation of all hip movements, with pain and spasm if movement is forced *Radiographic examination:* The earliest radiographic changes are usually present by the time advice is sought There is a slight decrease in the depth of the ossific nucleus of the femoral head, whereas the clear cartilage space is often increased in depth: in other words the bony nucleus seems to have shrunk within its surrounding bed of cartilage (Fig 223). The nucleus becomes denser than that of the normal side, and later it takes on a fragmented or granular appearance, areas of increased density being interspersed with areas of relative porosis. If weight-bearing is allowed to continue the nucleus is progressively flattened Eventually the texture of the bone returns to normal (Fig 224), but if flattening has occurred the femoral head is permanently deformed (Fig. 225).

Diagnosis. Perthes' disease is distinguished from tuberculous arthritis, which it resembles clinically, mainly by the radiographs.

FIG. 223

Perthes' disease of the left hip. Note the shrunken appearance of the bony nucleus of the femoral epiphysis, the corresponding increase in depth of the cartilage space, the patchy changes of density, and the suggestion of fragmentation

FIG. 224

Same patient as above, after two years' relief from weight-bearing. Shape of head virtually normal.

FIG. 225

Deformed head in an untreated case. Irregularity of the joint surface will eventually lead to osteoarthritis.

The normal erythrocyte sedimentation rate and blood count, and the good general health, are other distinguishing features.

Sequel. If the femoral head is deformed osteoarthritis always develops in later life—often between the ages of 30 and 50.

Treatment. The essential point in treatment is to prevent the deformation of the femoral head while it is in the softened state. The most satisfactory method is by rest in bed, with weight traction on the affected limb suspended in a Thomas splint. This treatment must be continued until the bone re-hardens—usually about two years after the onset of symptoms. The treatment is best carried out at a country orthopaedic hospital where facilities for education are available. If there are serious obstacles to prolonged treatment in hospital, protection of the hip by a weight-relieving patten-ended caliper is a reasonable though less reliable alternative.

SLIPPED UPPER FEMORAL EPIPHYSIS

(Adolescent coxa vara; epiphysial coxa vara)

This is an affection of late childhood in which the upper femoral epiphysis is displaced from its normal position upon the femoral neck. The displacement occurs at the epiphysial line.

Cause. This is unknown.

Pathology. The junction between the capital epiphysis and the neck of the femur loosens. With the downward pressure of weight-bearing and the upward pull of muscles on the femur the epiphysis is displaced from its normal position. Displacement is always downward and backwards, so that the epiphysis comes to lie at the back of the femoral neck (Fig. 226). The displacement usually occurs gradually, but occasionally a sudden displacement is caused by injury, such as a fall. Left undisturbed, the epiphysis fuses to the femoral neck in the abnormal position. The consequent deformity of the articular surface predisposes to the later development of osteoarthritis

Clinical features. *The patient is between 10 and 20 years of age.* In about half the cases there is evidence of an endocrine disturbance leading to a plump "fat-boy-of-Dickens" type of build; but in other examples the child is of normal development. In about half the cases both hips are affected, one after the other. Typically, there is a gradual onset of pain in the hip, with limp.

Rarely, these symptoms develop acutely after an injury. *On examination* the physical signs are characteristic, for there is selective limitation of certain hip movements, the other movements being full or even increased. The movements that are limited are flexion, abduction, and medial rotation. Lateral rotation and adduction are often increased, and the limb tends to lie in lateral rotation. *Radiographic examination:* Even a slight

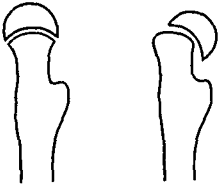

FIG. 226

Upper end of child's femur seen from the side. *Left*—Normal position of epiphysis. *Right*—Slipped epiphysis The displacement is always backwards and downwards.

displacement of the epiphysis is recognisable, provided good *lateral* radiographs are obtained. It must be stressed that a slight displacement is easily overlooked if antero-posterior films alone are examined (Fig. 230). Lateral radiographs are essential. In the lateral film the epiphysis is seen to be tilted over towards the back of the femoral neck,[1] the posterior " horn " being lower than the anterior (Figs 227 and 228).

Diagnosis. Slipped upper femoral epiphysis should be suspected in every patient of 10 to 20 who complains of pain in the hip. The characteristic clinical features, together with the radiographic evidence of epiphysial displacement, are conclusive. The condition is nevertheless often missed, simply because lateral radiographs are not obtained.

[1] Students often have difficulty in determining in lateral radiographs of the upper end of the femur which is the back and which is the front of the bone The key is the bony projection formed by the trochanters; this is always posterior.

FIG. 227 FIG. 228 FIG. 229

Figure 227—Slipped epiphysis: slight degree Figure 228—Slipped epiphysis: severe degree. Figure 229—After open reduction and fixation by a nail In studying slipped epiphysis lateral radiographs are essential.

FIG. 230

This radiograph shows how a slipped epiphysis may easily be overlooked if antero-posterior films alone are examined Same patient as shown in Figure 227 The only indications that the right femoral epiphysis is displaced are a slight reduction of its vertical depth and slight rounding of the upper corner of the femoral neck.

Sequel. If severe displacement is allowed to remain uncorrected osteoarthritis always develops in later life (Fig. 231).

Treatment. The treatment depends upon the degree of displacement.

Slight displacement. When displacement is slight (less than one centimetre as measured on the radiograph) (Fig. 227) the position can be accepted and all that is necessary is to prevent further displacement. This is achieved by driving a tri-flanged nail along the neck of the femur into the epiphysis.

Severe displacement. When displacement is severe (Fig. 228) the position cannot be accepted because of the certainty that painful osteoarthritis will develop in adult life. The position must therefore be improved. Three methods are available: manipulation, operative replacement of the epiphysis at the site of slipping, and compensatory osteotomy at a lower level

FIG. 231
Osteoarthritis developing twenty years after uncorrected slipped epiphysis

Manipulation is seldom successful; it is worth trying only in cases of recent displacement caused by injury.

Operative replacement of the epiphysis with fixation by a nail is recommended for cases in which symptoms have been present for less than three months. At such a relatively early stage the operation can restore the hip virtually to normal (Fig. 229) Its disadvantage is that it may impair the blood supply of the epiphysis, thereby leading to avascular necrosis of the femoral head and precipitating the onset of osteoarthritis.

Compensatory osteotomy is recommended for cases in which symptoms have been present for more than three months By that time adaptive changes have occurred in the bone and they preclude the restoration of a normal joint by operative replacement

of the epiphysis. The osteotomy is done just below the trochanteric level. A wedge of bone is removed so that the shaft of the femur is angled into flexion and abduction relative to the upper fragment; at the same time the shaft is rotated medially. The operation thus compensates for the downward and backward tilting of the epiphysis; correction should be sufficient to bring the epiphysis once more into the weight-transmitting segment of the acetabulum. The operation entails no risk of damage to the blood supply of the femoral head, but it leaves the articular surface of the upper end of the femur deformed, and therefore does not altogether remove the risk of secondary osteoarthritis in later years.

EXTRA-ARTICULAR DISORDERS IN THE REGION OF THE HIP

COXA VARA

The general term coxa vara includes any condition in which the neck-shaft angle of the femur is less than the normal of about 125 degrees. The angle is sometimes reduced to 90 degrees or less The deformity is caused mechanically by the stress of body weight acting upon a femur that is defective or abnormally weak.

Causes. The most important causes of coxa vara are: 1) *Congenital.* Part of the femoral neck remains as unossified cartilage, which gradually bends during childhood (congenital coxa vara; infantile coxa vara). This type is uncommon. 2) *Slipped upper femoral epiphysis* (epiphysial coxa vara). This was described on page 312 3) *Fracture.* Coxa vara is common after fractures in the trochanteric region with mal-union, and in ununited fractures of the neck of the femur. 4) *Softening of bone,* in general affections such as rickets, osteomalacia, or parathyroid osteodystrophy

Effects. Coxa vara leads to true shortening of the limb Approximation of the greater trochanter to the ilium impairs the efficiency of the hip abductors, leading in severe cases to a Trendelenburg " dip " and consequent limp (p 291).

Treatment. The treatment is mainly that of the underlying condition. In appropriate cases the neck-shaft angle can be corrected by osteotomy just below the greater trochanter.

OSTEOMYELITIS OF THE UPPER FEMORAL METAPHYSIS

Osteomyelitis of the femur will be described with disorders of the thigh in the next chapter. It is sufficient to mention here that acute pyogenic infection of the upper end of the femur, which is not uncommon in children, may spread directly to involve the hip, for the metaphysis is enclosed within the capsule of the joint.

EXTRA-ARTICULAR TUBERCULOSIS OF BONE

Isolated tuberculous infection of bone, as distinct from infection spreading from a joint, is uncommon. Nevertheless it is well recognised both in the upper end of the femur and in the os innominatum. The lesion occurs as a rounded cavity within the bone, containing tuberculous pus or granulation tissue. There is a deep diffuse pain, but joint movements are unimpaired.

Radiographs reveal a rounded zone of osteoporosis, without any reaction in the surrounding bone (Fig. 41, p. 62). In the absence of efficient treatment most of these lesions eventually spread to involve the hip, with the usual features of tuberculous arthritis

Treatment. When the lesion is accessible it should be drained by drilling the overlying bone after a preliminary course of systemic chemotherapy (streptomycin, para-amino-salicylic acid, and isonicotinic acid hydrazide) The incision must be kept well away from the joint.

TUBERCULOSIS OF THE TROCHANTERIC BURSA

The extensive bursa between the greater trochanter and the gluteal aponeurosis is occasionally the site of tuberculous infection.

Pathology. The tubercle bacilli presumably reach the bursa through the blood stream from a focus elsewhere. There is a chronic inflammatory reaction in the walls of the bursa, usually with the formation of a tuberculous abscess. The abscess may burst through the skin, giving rise to a chronic discharging sinus Later, the surface of the greater trochanter is sometimes eroded

Clinical features. The patient is usually a young adult. He complains of a swelling in the trochanteric region, with local

discomfort and sometimes a persistent discharge of pus *On examination* the trochanteric area is thickened, warmer than normal, and perhaps reddened. Often there is a palpable abscess or a discharging sinus. Movements of the hip are not impaired. *Radiographs* typically show no abnormality, but in long-standing cases there is sometimes a superficial roughening or erosion of the lateral aspect of the greater trochanter. *Investigations:* The erythrocyte sedimentation rate is increased. Biopsy of the walls of the bursa shows the typical histological features of tuberculosis.

Complications. Secondary involvement of the hip joint, with the usual features of tuberculous arthritis, has hitherto been a common late complication. Probably its frequency will be reduced by effective treatment of the primary bursitis.

Treatment. This should be carried out at a country orthopaedic hospital. After a three months' preliminary period of rest and systemic chemotherapy, with aspiration as required, the bursa should be excised entire.

SNAPPING HIP

Snapping hip is a harmless condition in which a distinct "snap" is heard and felt on certain movements of the joint. It does not denote any underlying injury or disease and it is of no practical significance. The snap is attributed to slipping of a tendinous aponeurosis—probably that of the gluteus maximus—over the bony prominence of the greater trochanter It is heard easily when the patient flexes the hip actively, but it is not reproduced by passive movement with the muscles relaxed Treatment is not required

EXTRINSIC DISORDERS SIMULATING DISEASE OF THE HIP

As has already been mentioned, it frequently happens that a patient complains of symptoms in the region of the hip or thigh when in fact they arise at a distance The conditions that may confuse the diagnosis in this way fall into two main groups: 1) disorders of the spine or sacro-iliac joints, and 2) disorders of the abdomen or pelvis

DISORDERS OF THE SPINE AND SACRO-ILIAC JOINTS

PROLAPSED INTERVERTEBRAL DISC

The pain of a prolapsed or strained lumbar intervertebral disc is often referred to the gluteal region or lateral aspect of the thigh: indeed this is the commonest clinical feature in cases of intervertebral disc injury of slight or moderate degree. The patient himself, and often his doctor, may ascribe the symptoms to a lesion of the hip. But the history is unlike that of a hip affection. On examination other evidence of a spinal disorder will usually be found, whereas the hip itself is normal clinically and radiographically.

SACRO-ILIAC ARTHRITIS

The pain caused by arthritis of a sacro-iliac joint—whether it be tuberculous, pyogenic, or the early stages of ankylosing spondylitis—spreads diffusely over the gluteal area and may simulate an affection of the hip. Mistakes should be prevented by careful clinical examination and by routine radiography of the whole pelvis in cases of alleged hip complaints.

DISORDERS OF THE ABDOMEN AND PELVIS

PELVIC OR LOWER ABDOMINAL INFLAMMATION

Inflammation involving the side wall of the pelvis may mimic a hip lesion very closely; indeed even experienced surgeons have been deceived The condition responsible is usually a subacute suppurative lesion such as a deep peri-appendicular abscess or a pyosalpinx. The hip symptoms arise partly from irritation of the obturator nerve, causing referred pain in the thigh, and partly from irritative spasm of the hip muscles that have their origin within the abdomen or pelvis—namely the psoas, iliacus, pyriformis, and obturator internus. The muscle spasm may cause marked restriction of hip movements, with pain if movement is forced. Differentiation from an intrinsic lesion of the hip depends upon a careful history and a complete physical examination, including an investigation of the abdomen and pelvis. It is always important to bear in mind the possibility of abdominal or pelvic inflammation when the nature of an alleged hip complaint remains in doubt after clinical and radiographic examination of the joint.

CHAPTER NINE

The Thigh and Knee

THE knee depends for its stability upon its four main ligaments and upon the quadriceps muscle. The importance of the quadriceps cannot be over-emphasised. So efficiently can a powerful quadriceps control the knee that it can maintain stability despite considerable laxity of the ligaments. In many injuries and diseases of the knee the quadriceps wastes strikingly; and to some extent the condition of the muscle is an index of the state of the knee. If it is wasted it is highly probable that there is an abnormality within the joint.

Apart from its vulnerability to injury, the knee is also particularly prone to almost every kind of arthritis. Moreover, it is the joint most commonly affected by osteochondritis dissecans and intra-articular loose-body formation

The region of the knee is the zone of most active bone growth in the lower limb (contrast the upper limb, where most growth occurs towards the shoulder and wrist). Perhaps partly for this reason the metaphyses near the knee are common sites of osteomyelitis and of primary malignant bone tumours.

The knee is, in fact, a region where nearly every kind of orthopaedic disorder may be represented.

SPECIAL POINTS IN THE INVESTIGATION OF THIGH AND KNEE COMPLAINTS

History

The history is of particular importance in the diagnosis of disorders of the knee. In cases of torn semilunar cartilage, for instance, the history is often the most important factor in the diagnosis. When there has been a previous injury to the knee, the exact sequence of events at the time of the injury and afterwards must be ascertained. Enquiry is made into the mechanism of the injury; what the patient was doing at the time, whether he was able to carry on afterwards. Was he able to finish the game? If not, was he carried from the field or was he able to walk? How soon after the injury did the knee swell? Was

he able to straighten the knee fully? If not, how did he get it straight? Was he able to bend it? These and many other details must be elicited by careful questioning because they are so important in building up a picture of what exactly happened to the knee. Caution is necessary in accepting the patient's story of "*locking*" at its face value. Many patients speak of "locking" when the knee simply feels stiff and

TABLE X

ROUTINE CLINICAL EXAMINATION IN SUSPECTED DISORDERS OF THE THIGH AND KNEE

1. LOCAL EXAMINATION OF THE THIGH AND KNEE

Inspection
- Bone contours and alignment
- Soft-tissue contours
- Colour and texture of skin
- Scars or sinuses

Palpation
- Skin temperature
- Bone contours
- Soft-tissue contours
- Local tenderness

Measurement of thigh girth
- Comparative measurements at precisely the same level in each limb. (Note particularly the bulk of the quadriceps muscle)

Movements (active and passive, against normal knee for comparison)
- Flexion
- Extension
- ? Pain on movement
- ? Crepitation on movement

Power (tested against resistance of examiner)
- Flexion
- Extension

Stability
- Medial ligament
- Lateral ligament
- Anterior cruciate ligament
- Posterior cruciate ligament

Rotation tests (McMurray)
- (Of value only when a torn cartilage is suspected)

Stance and gait

2. EXAMINATION OF POTENTIAL EXTRINSIC SOURCES OF THIGH OR KNEE SYMPTOMS

This is important if a satisfactory explanation for the symptoms is not found on local examination. The investigation should include 1) the spine, and 2) the hip

3. GENERAL EXAMINATION

General survey of other parts of the body. The local symptoms may be only one manifestation of a widespread disease.

painful or when it causes momentary jabs of pain on movement. True locking from a torn cartilage means simply that the knee cannot be *straightened fully*; it can usually be flexed freely. In locking from a loose body within the joint the knee may be jammed so that *it will* neither flex nor extend, but this is rather uncommon and the knee usually unlocks itself after an interval.

Exposure

For proper examination the whole length of the limb must be uncovered. In the case of male patients it is essential that the trousers (and long pants, if worn) be removed. It is impossible to examine a knee adequately when the thigh is half covered by tightly rolled trousers or underpants. The sound knee must also be exposed for comparison. The patient must be recumbent upon a couch—not just sitting with his foot on a stool.

Steps in Clinical Examination

A suggested routine for clinical examination of the thigh and knee is summarised in Table X.

Determining the Cause of a Diffuse Joint Swelling

The knee exemplifies better than any other joint the different types of diffuse articular swelling. That the joint is in fact swollen should be obvious from inspection: comparison of the two knees will show that the concavities normally present at each side of the patella have been filled out on the affected side.

A diffuse swelling of the knee can arise only from three fundamental causes: 1) thickening of bone, 2) fluid within the joint; and 3) thickening of the synovial membrane (in practice it is found that this is the only soft tissue about the knee that swells appreciably). Determination of the particular cause or combination of causes in a given case depends entirely on careful palpation.

Thickening of bone Thickening of bone is detected without difficulty by deep palpation if the affected side is compared with the normal. There may be a general enlargement, caused perhaps by a bone infection or by an expanding tumour or cyst; or there may be simply a local prominence, caused usually by osteophytes at the joint margin or by an exostosis.

Fluid within the joint. With practice, it is easy to detect even a small fluid effusion. It must be emphasised, however, that the widely used "patellar tap" test is unreliable The test is negative in the presence of fluid in two circumstances: first, when there is insufficient fluid to raise the patella away from the femur; and secondly, when there is a tense effusion. If used at all, the "patellar tap" should be used only as a supplementary test. A fluid effusion is best detected by the fluctuation test. The palm of one hand is placed upon the thigh immediately above the patella—that is, over the suprapatellar pouch.

The other hand is placed over the front of the joint, with the thumb and index finger just beyond the margins of the patella (Fig. 232). Pressure of the upper hand upon the suprapatellar pouch drives fluid from the pouch into the main joint cavity, where it bulges the capsule at each side of the patella and imparts an easily detectable hydraulic impulse to the finger and thumb of the lower hand. Conversely, by pressure of this finger and thumb the fluid can be driven back into the suprapatellar pouch, the hydraulic impulse being clearly received by the upper hand. In this way an unmistakable sense of fluctuation can be elicited between the two hands.

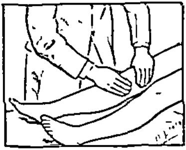

FIG. 232

Fluctuation test for intra-articular fluid. Fluctuation is elicited between the two hands. One hand compresses the suprapatellar pouch, while the thumb and fingers of the other receive the impulse at either side of the patella.

Distinction between effusions of blood, serous fluid, and pus is made partly from the history, partly from the clinical examination. An effusion of blood (haemarthrosis) appears within a few hours of injury and rapidly becomes tense. An effusion of clear fluid develops slowly (twelve to twenty-four hours) and is never so tense as a blood effusion. An effusion of pus is associated with general illness and pyrexia.

Thickening of the synovial membrane. A thickened synovial membrane is always a prominent feature of chronic inflammatory arthritis. The thickening is often most obvious above the patella, where the reduplicated membrane forms the suprapatellar pouch. It has a characteristic " boggy " feel on palpation, rather as if a sheet of sponge rubber had been placed between the skin and the underlying bone. It is worth noting that since it is highly vascular a thickened synovial membrane is always associated with increased warmth of the overlying skin.

Movements

Accurate assessment of the range of movement is particularly important in the knee, because even a slight impairment of movement is significant. It is important to note also whether movement is painful and whether it is accompanied by crepitation.

Flexion. The normal range varies with the build of the patient. Thin patients can flex more than fat patients—usually enough to bring the heel in contact with the buttock. The range of the sound knee must be taken as the normal for the individual.

Extension. There is a fairly wide variation in the normal range: it is wrong to accept 180 degrees as the normal. In general, most normal women can hyperextend their knees whereas some normal men cannot extend quite to 180 degrees. It is important to detect even a slight

impairment of knee extension: therefore the range on the sound side must be taken as the yardstick of normal extension and a very careful comparison must be made between the two sides.

Tests for Stability

The integrity of each of the four major ligaments is tested in turn

Testing the medial and lateral ligaments. For this test the joint must be in full extension and the quadriceps must be relaxed. Normally the ligaments are taut only when the knee is fully extended. if the knee is slightly flexed the ligaments will be slack even though they are undamaged. *Technique:* Support the limb by a hand gripping the ankle region. Instruct the patient to relax the muscles so that the limb drops back into full extension. See that the quadriceps muscle is relaxed. Using the free hand as a fulcrum at the side of the knee, apply

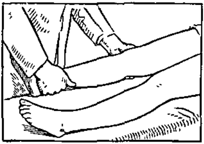

FIG 233
Testing the medial ligament

ament (Fig. 233)

If the ligament

If the ligament

is strained but not torn, the joint will remain stable but stress applied to the ligament will cause pain.

Testing the anterior and posterior cruciate ligaments. The knee must be flexed 90 degrees, the foot must be fixed on the couch, and the quadriceps must be relaxed. The *anterior* cruciate ligament prevents *anterior* glide of the tibia on the femur. the *posterior* cruciate ligament prevents *posterior* glide. *Technique.* The patient's knee being flexed to a right angle and the foot placed firmly on the couch, sit lightly on the foot to prevent it from sliding (Fig. 234). With the interlocked fingers of the two hands form a sling behind the upper end of the tibia, and clasp the sides of the leg between the thenar eminences. Place the tips of the thumbs one upon each femoral condyle. Alternately pull and push the upper end of the tibia to determine the amount of antero-posterior movement Normally there is an antero-posterior glide of up to a quarter of an inch; but since the normal is variable it is wise to use the patient's sound limb for comparison. Excessive glide in one or other direction indicates damage to the corresponding cruciate ligament.

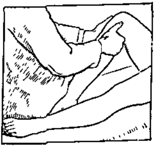

FIG 234
Testing the cruciate ligaments The patient's foot is steadied by sitting upon it. Pulling the tibia forwards tenses the anterior ligament, pushing it backwards tenses the posterior ligament

The Rotation Test for Pedunculated Cartilage Tag.

In this manoeuvre, often known as McMurray's test, the tibia is rotated upon the femur with the knee in various positions of flexion and extension. Its object is to demonstrate mobile pedunculated tags of cartilage, and the test is important only when a torn semilunar cartilage is suspected. By manoeuvring the knee it is hoped to cause the pedunculated tag, if present, to become temporarily jammed between the bone ends, so that a loud click or " clonk " will be produced when the tag is dise [illegible] The limb is grasped fir [illegible] as control of rotation of [illegible] the knee. The free hand is placed over the knee to feel for clicks. The rotation tests are started with the knee fully flexed and repeated with the knee in progressively less flexed positions. The first test: The knee being flexed fully, rotate the tibia laterally upon the femur to the full extent. Now straighten the knee slowly while the rotation is maintained: listen and feel for a click. Repeat the test in exactly the same way but with the tibia rotated medially instead of laterally. Then repeat, alternately with lateral and medial rotation, with the knee in less degrees of flexion—for example, at 60 degrees, 75 degrees, 90 degrees, 105 degrees, and 120 degrees. A loud click, distinct from the normal patellar click and usually associated with pain, suggests a tag tear (not a " bucket-handle " tear) of a semilunar cartilage. *Caution:* Loud clicks can often be produced in normal knees. Most of them arise from movements of the patella, and they are not accompanied by pain. Discretion must be used in interpreting a click as an abnormal finding.

Extrinsic Causes of Pain in the Thigh and Knee

In most cases symptoms felt in the knee have their origin locally in or near the joint. But there are exceptions that may trap the unwary. Most important, pain in the knee may be the predominant feature of an arthritic hip. Less commonly a sciatic pain, perhaps from a prolapsed intervertebral disc, has its greatest intensity at the level of the knee. In the investigation of pain in the knee we must therefore recognise the possibility that it may be referred from the spine or hip, and extend the examination to those regions if a satisfactory explanation for the trouble is not found on local examination.

Radiographic Examination

In the routine radiographic examination of the knee plain antero-posterior and lateral films are sufficient. They should include a reasonable length of the femur and of the tibia and fibula. Tangential projections of the femoral condyles with the knee flexed are sometimes helpful, especially when osteochondritis dissecans is suspected. When it is suspected that the knee symptoms might be referred from a lesion of the hip or spine appropriate radiographs of those regions should be obtained.

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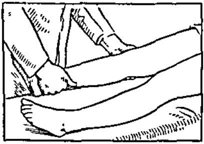

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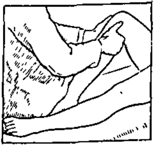

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CLASSIFICATION OF DISORDERS OF THE THIGH AND KNEE

DISORDERS OF THE THIGH

INFECTIONS

- Acute osteomyelitis
- Chronic osteomyelitis
- Syphilitic infection

TUMOURS

- Benign bone tumours
- Malignant bone tumours

ARTICULAR DISORDERS OF THE KNEE

DEFORMITIES

- Genu varum and genu valgum

ARTHRITIS

- Pyogenic arthritis
- Rheumatoid arthritis
- Tuberculous arthritis
- Osteoarthritis
- Haemophilic arthritis
- Charcot's osteoarthropathy
- Clutton's joints
- Chondromalacia of the patella

MECHANICAL DISORDERS

- Tears of the semilunar cartilages
- Cysts of the semilunar cartilages
- Discoid lateral semilunar cartilage
- Osteochondritis dissecans
- Intra-articular loose bodies
- Recurrent dislocation of the patella

EXTRA-ARTICULAR DISORDERS IN THE REGION OF THE KNEE

INJURIES

- Rupture of the quadriceps apparatus

CYSTIC SWELLINGS

Infrapatellar bursitis
Baker's cyst
Semimembranosus bursitis

POST-TRAUMATIC OSSIFICATION

Pellegrini-Stieda's disease of the medial femoral condyle

OSTEOCHONDRITIS

Osgood-Schlatter's disease of the tibial tubercle

DISORDERS OF THE THIGH

ACUTE OSTEOMYELITIS

(General description of acute osteomyelitis, p. 54.)

The femur is one of the bones most commonly affected by pyogenic osteomyelitis.

Pathology. In most cases the infection is carried to the femur through the blood stream (haematogenous osteomyelitis). Occasionally it is introduced through an external wound, especially in cases of compound fracture, but such injuries are uncommon except in time of war.

In the haematogenous type the infection begins in the metaphysis at one end of the bone, the lower metaphysis being affected more often than the upper. The upper metaphysis of the femur is wholly within the capsule of the hip joint, and the lower metaphysis is partly within the capsule of the knee joint. Consequently infection may spread directly from a metaphysial focus to involve the adjacent joint, or vice versa.

Clinical features. In the typical haematogenous infection the patient is an infant or child. There is constitutional illness, with pyrexia. There is severe pain at the site of infection—usually near the lower end of the femur. *On examination* there is marked local tenderness on palpation. An area of induration develops and later, in neglected cases, an abscess forms beneath the

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periosteum and may extend into the surrounding soft tissues. In the absence of complicating pyogenic arthritis the adjacent joint remains freely movable, though it may contain an effusion of clear fluid (" sympathetic " effusion). If the infection spreads to the joint the features of pyogenic arthritis will be superimposed upon those of osteomyelitis. *Radiographs* show no abnormality in the early acute stage. Later, there are local osteoporosis and new bone formation beneath the stripped-up periosteum (Fig. 32, p. 57). *Investigations:* There is a marked polymorphonuclear leucocytosis.

In acute osteomyelitis complicating a compound fracture the clinical picture is different. The patient is usually an adult. The temperature fails to settle after the primary treatment of the wound, and pus collects in the tissue planes or pours from the wound. Any part of the bone may be affected. *Radiographs* show the fracture, but at first they give no indication that the bone is infected.

Treatment. This is the same as for acute osteomyelitis elsewhere. In acute haematogenous osteomyelitis the mainstay of treatment is chemotherapy by penicillin or other appropriate antibiotic, which should be begun at the earliest possible moment. If an abscess forms it must be drained immediately. A careful watch should be kept for evidence of involvement of the hip or knee.

In osteomyelitis complicating an open fracture the essential principle of treatment is to secure free drainage.

CHRONIC OSTEOMYELITIS

(General description of chronic osteomyelitis, p. 59.)

Chronic pyogenic osteomyelitis is nearly always a sequel to acute osteomyelitis that has been neglected or has responded poorly to treatment.

Pathology. As with the acute disease, the lower end of the femur is affected more often than the upper; but in many cases the infection spreads to involve a large part or even the whole of the shaft. The bone is thickened irregularly, and whereas parts of it are sclerotic it is often honeycombed with small or large cavities containing pus or granulation tissue. There may be an intermittent or a persistent discharge of pus from a sinus. In many

cases there is bony or fibrous ankylosis of the adjacent joint (hip or knee), indicating that the original acute infection had spread to the joint (Fig. 35, p. 58).

Clinical features. These vary from case to case. There may simply be a chronic discharge from a sinus, or there may be recurrent "flares" of infection with intervening periods of quiescence. *Radiographs* show the irregular thickening of the femoral shaft, with patchy areas of sclerosis and cavitation. Sometimes a dense sequestrum is seen lying loose within a cavity.

Treatment. This is the same as for chronic osteomyelitis elsewhere. Recurrent "flares" of infection will often subside with systemic chemotherapy and rest alone. If there is persistent profuse discharge from a sinus operation is required. The objects are to remove all dead bone and to eliminate bone cavities by de-roofing them and allowing the soft tissues to fall in.

SYPHILITIC INFECTION

(General description of syphilis of bone, p. 64.)

Syphilitic disease of bone is now rare in Great Britain, but it is still seen occasionally and the possibility of its occurrence should be borne in mind. The femur is one of the commonest sites of syphilitic gumma and of diffuse syphilitic osteoperiostitis.

Clinical features. The complaint is of a gradually enlarging swelling, with moderate pain The swelling feels hard or firm, and it may reach large proportions. *Radiographs* may show a diffuse thickening of the femoral shaft, sometimes with a more localised area of destruction.

Diagnosis. A syphilitic swelling is easily mistaken for a tumour unless the possibility of syphilis is considered. The Wassermann and Kahn tests are positive. Rapid improvement after the administration of penicillin and other anti-syphilis drugs is of important diagnostic significance.

BONE TUMOURS IN THE THIGH

The femur is one of the commonest sites of the important bone tumours.

BENIGN TUMOURS

(General description of benign bone tumours, p. 66.)

Of the four main types of benign bone tumour—osteoma, chondroma, osteochondroma, and osteoclastoma — only the osteoclastoma requires further consideration here.

Osteoclastoma (Giant-cell tumour)

The lower end of the femur is a particularly frequent site for this tumour. The upper end is affected much less often The tumour usually arises in young adults. It begins in what was the metaphysial region, but since the epiphysis is fused there is no obstacle to its spread into the articular end of the bone (Fig. 50, p. 69). The bone is gradually " expanded," the cortex becoming very thin. Pathological fracture may occur. Though classed with the benign tumours, an osteoclastoma tends to recur after removal and in some cases it behaves like a sarcoma, metastasising through the blood stream.

Treatment. It is impracticable to carry out a complete local excision of an osteoclastoma of the femur without interfering seriously with the adjacent joint. Therefore reliance is usually placed upon thorough curettage of the tumour, the resulting cavity being filled with bone chips. But if biopsy casts doubt upon the innocence of the tumour wide local excision, with pre-operative and post-operative radiotherapy, should be advised.

MALIGNANT TUMOURS

(General description of malignant bone tumours, p 70.)

The femur is a common site for all of the four main types of malignant bone tumour.

Osteogenic Sarcoma

This is usually a tumour of childhood , when it occurs in patients beyond middle age it is often a complication of Paget's disease. Typically it arises in the metaphysis of a long bone, and the lower metaphysis of the femur is a favourite site. The tumour is highly malignant, and despite treatment it usually causes death from pulmonary metastases.

Ewing's Tumour

This also occurs mainly in children. Unlike most bone tumours, it affects the shaft of the bone, which it expands in a fusiform manner. Over it, layer upon layer of new bone is laid down, giving a typical "onion-peel" appearance as seen in the radiograph. Although the tumour responds dramatically to deep x-ray therapy for a while it is ultimately fatal from blood-borne metastases.

Multiple Myeloma

This is a tumour of later life. There are multiple tumour foci scattered through several bones, especially those containing abundant red marrow. The upper half of the femur is a favourite site (Fig 53, p. 71). The tumour responds temporarily to deep x-ray therapy but it is eventually fatal.

Metastatic (Secondary) Tumours

Metastatic tumours of bone are very much more common than any of the primary malignant tumours. The femur is commonly affected, especially in its proximal half (Fig. 235).

Pathology. The tumours that metastasise most readily to bone are carcinomas of the lung, breast, prostate, kidney, and thyroid The bone structure is destroyed by the tumour (except in the occasional osteoblastic or bone-forming metastasis from the prostate). Pathological fracture is common

Clinical features. Pain is the predominant symptom, but sometimes the tumour causes little disturbance until a pathological fracture occurs

Treatment. Deep x-ray therapy often affords temporary relief. Hormone therapy is worth trying in metastases from the breast

Fig. 235
Metastatic tumour in femur—a common site The primary tumour was in the lung

and prostate, and in some cases adrenalectomy may be worth considering. Most pathological fractures of the femur lend themselves well to internal fixation with a nail-plate or a long intramedullary nail. This facilitates nursing and greatly increases the patient's comfort, for external splints can be dispensed with.

ARTICULAR DISORDERS OF THE KNEE

GENU VARUM AND GENU VALGUM

The common childhood deformities of the knee are seldom important, because they tend to undergo spontaneous correction. But the possibility of underlying disease, such as rickets, must

FIG. 236

Genu varum Slight bowing is common in infants but it is usually corrected spontaneously as the child grows.

FIG. 237

Genu valgum. Deformity of this degree in young children is usually corrected spontaneously during growth.

always be remembered The two deformities to be considered are genu varum (bow leg) and genu valgum (knock knee).

GENU VARUM

The knee is bowed outwards (Fig. 236). A mild degree of this deformity is so common as to be almost normal in children of 1 to 3 years. It requires no treatment unless it persists into later childhood. Care should be taken to exclude the possibility of rickets.

GENU VALGUM

The knee is angled inwards, the tibia being abducted in relation to the femur. With the knees straight, the medial malleoli cannot be brought into contact (Fig. 237). The deformity is common in children of 3 to 5 years. In the absence of underlying bone disease it usually corrects itself spontaneously in the course of years.

Treatment. In early childhood treatment is unnecessary, but it is common practice to fit a wedge, base medially and $\frac{1}{8}$ to $\frac{3}{16}$ inch deep, to the heel of the shoe to shift the line of weight-bearing medially (Fig. 277, p. 387). Severe genu valgum persisting after the age of 10 requires active treatment. In growing children two methods of correction are available. One is to retard the growth at the medial side of the epiphysial cartilage of femur or tibia by bridging epiphysis to diaphysis with metal staples (epiphysiodesis). The other and more certain method is by supracondylar osteotomy of the femur, with excision of a suitable wedge from the medial side. After the cessation of epiphysial growth genu valgum can be corrected only by osteotomy.

PYOGENIC ARTHRITIS OF THE KNEE

(General description of pyogenic arthritis, p. 31.)

Pyogenic arthritis is commoner in the knee than in most other joints, partly because the knee is so exposed to injury and partly because of the close relationship of the joint cavity to the lower metaphysis of the femur, which is one of the commonest sites of acute osteomyelitis. The organism usually responsible is the staphylococcus or streptococcus, less often the pneumococcus or gonococcus

Pathology. The organisms reach the joint through the blood stream, through a penetrating wound, or from an adjacent focus of osteomyelitis Unless the infection can be cut short by prompt treatment the joint is usually destroyed, and the final outcome is a fibrous or bony ankylosis.

Clinical features. The onset is acute or subacute. There is constitutional illness with pain, swelling, and loss of function of the knee. *On examination* there is pyrexia. The knee is abnormally warm and distended with fluid. Movements are greatly restricted by pain and protective muscle spasm. A penetrating wound or a septic focus elsewhere may be apparent. *Radiographs* show no abnormality in the early stages. *Investigations :* There is a polymorphonuclear leucocytosis and a raised erythrocyte sedimentation rate. Aspiration of the joint yields turbid fluid or pus, from which the causative organism can usually be identified.

Treatment. *Constitutional treatment* is by rest and chemotherapy. Whenever possible the causative organism must be identified and its sensitivity to antibiotic drugs determined, so that the most effective drug can be given. *Local treatment* is by rest for the joint (usually in a Thomas's splint or plaster) and drainage of pus by daily aspiration. After each aspiration a solution of the appropriate antibiotic drug is injected into the joint. Rest is continued until the infection has been overcome ; thereafter active movements are encouraged.

RHEUMATOID ARTHRITIS OF THE KNEE

(General description of rheumatoid arthritis, p 34)

The knees are among the joints most frequently affected by rheumatoid arthritis, and they often suffer severe permanent disability. As in other joints, the most striking change is a chronic non-bacterial inflammation of the synovial membrane, with later a tendency to softening and erosion of the articular cartilage.

Clinical features. Both knees are often affected simultaneously with several other joints. The knees are swollen and painful The pain is worst when activity is resumed after resting *On examination* the knee is swollen from synovial thickening. The overlying skin is warmer than normal Movements are impaired, and painful if forced The thigh muscles are wasted. *Radiographic examination* At first there are no changes. Later there is diffuse osteoporosis in the area of the joint. In long-established cases destruction of articular cartilage leads to narrowing of the cartilage space (Fig 238), and there may be local clear-cut erosions of bone *Investigations* · The erythrocyte sedimentation rate is raised during the active phase

urse and prognosis. The inflammation dies down after . nths or years, but the knee is seldom restored to normal. The nt surfaces are usually damaged and ar out sooner than those of a normal nt. Thus osteoarthritis is liable to superimposed upon the original eumatoid condition.

FIG. 238
Long-established rheumatoid arthritis of the knee. The bone is porotic and the cartilage space is narrowed.

eatment. In the active stage the atment is that for rheumatoid arthritis general. It includes constitutional easures to improve the general health, d systemic drugs such as gold salts, pirin, phenyl-butazone, or possibly rtisone or ACTH. Local treatment r the knees depends upon the severity the inflammatory reaction. If it is vere, rest in bed or even temporary nmobilisation in plaster is advisable. Vhen it is moderate or slight, activity ithin the limits of pain is encouraged. hysical treatment is worth a trial. he most effective methods are exercises to preserve muscle ower and joint movement, and local heat in the form of short-ave diathermy.

)perative Treatment. In severe cases with destruction of joint artilage, disabling pain, and a tendency to fixed flexion deformity peration may be required. Arthroplasty has been attempted, but t is unreliable Arthrodesis is therefore the method of choice. This gives excellent results when the disease is confined to one knee, and the improvement of function is usually gratifying even when both knees have to be fused.

TUBERCULOUS ARTHRITIS OF THE KNEE

(General description of tuberculous arthritis, p. 37)

After the hip, the knee is the joint most commonly affected by tuberculosis.

Clinical features. Children and young adults are most commonly affected. There is complaint of pain in the knee and a limp is noticed. *On examination* the knee is diffusely swollen

from thickening of the synovial membrane. The overlying skin is warmer than normal. Movements are limited, and if forced they provoke pain and muscle spasm. The thigh muscles are markedly wasted. An abscess or sinus is sometimes apparent. A

FIG. 239

Early tuberculous arthritis, with the normal knee shown for comparison. Note, on the affected side, the osteoporosis and the narrowed cartilage space. Though only slight, these changes are of great diagnostic importance when considered together with the clinical findings. In this case the disease did not progress to the stage of bone erosion, but its tuberculous nature was confirmed by biopsy.

tuberculous lesion may be discovered elsewhere. *Radiographic examination:* The earliest change is diffuse osteoporosis throughout the area of the knee (Fig. 239). Later, unless the disease is arrested, there are narrowing of the cartilage space and erosion of the underlying bone. *Investigations:* The erythrocyte sedimentation rate is increased The Mantoux test is positive. Biopsy of the synovial membrane (and sometimes of the inguinal glands) reveals the histological features of tuberculosis.

Course. There is a tendency towards slow healing if conditions are favourable. With early treatment a useful joint or even a normal joint can be saved in a proportion of cases, especially in

children. But in many cases the joint is destroyed and bony fusion secured by operation is the most satisfactory end-result.
Treatment. The patient should be admitted to a country orthopaedic hospital. *Constitutional treatment :* This is by rest and systemic chemotherapy (streptomycin, para-amino-salicylic acid and isonicotinic acid hydrazide). The drugs are continued for six months if well tolerated. *Local treatment :* Initial treatment is always by rest in a splint or plaster for the first six months (that is, during the course of chemotherapy). The subsequent management depends upon the response to treatment and the state of the joint at the end of the first six months' treatment. If the articular cartilage and bone are still intact, if the general health is good and the local signs have subsided, and if the erythrocyte sedimentation rate has shown a steady improvement, there is a good prospect that the disease has been aborted. In that event active joint movements are encouraged and walking is gradually resumed.

On the other hand, if the review at the end of the initial six months' treatment shows that the disease is still active and that articular cartilage or bone has been destroyed, the joint is " written off " as irrecoverable and a sound bony fusion becomes the ultimate aim. To this end immobilisation is continued for many months until the disease becomes quiescent. Arthrodesis is then undertaken if the patient is over 12 years old. In children under that age the joint is protected in a walking caliper or splint until the age of 12 is reached.

OSTEOARTHRITIS OF THE KNEE

(General description of osteoarthritis, p. 41.)

The knee is affected by osteoarthritis more often than any other joint. The condition is particularly common in fat women.
Cause. It is caused by wear and tear : but nearly always some factor is present that has caused the joint to wear out sooner than usual. Overweight is the commonest factor : for some reason it seems to impose a harmful stress upon the knee whereas it does not adversely affect the hip or ankle. Other important predisposing factors are : previous fracture causing irregularity of the joint surfaces ; previous disease with damage to the joint surfaces (especially old rheumatoid arthritis) ; and mal-alignment of the femur on the tibia (as in long-established bow leg).

from thickening of the synovial membrane. The overlying skin is warmer than normal. Movements are limited, and if forced they provoke pain and muscle spasm. The thigh muscles are markedly wasted. An abscess or sinus is sometimes apparent. A

FIG 239

Early tuberculous arthritis, with the normal knee shown for comparison. Note, on the affected side, the osteoporosis and the narrowed cartilage space Though only slight, these changes are of great diagnostic importance when considered together with the clinical findings. In this case the disease did not progress to the stage of bone erosion, but its tuberculous nature was confirmed by biopsy

tuberculous lesion may be discovered elsewhere. *Radiographic examination :* The earliest change is diffuse osteoporosis throughout the area of the knee (Fig 239) Later, unless the disease is arrested, there are narrowing of the cartilage space and erosion of the underlying bone. *Investigations* · The erythrocyte sedimentation rate is increased. The Mantoux test is positive. Biopsy of the synovial membrane (and sometimes of the inguinal glands) reveals the histological features of tuberculosis.

Course. There is a tendency towards slow healing if conditions are favourable. With early treatment a useful joint or even a normal joint can be saved in a proportion of cases, especially in

Treatment. Conservative treatment is usually effective in relieving the symptoms, although the structural changes in the joint are clearly irreversible. The most effective treatment is by physiotherapy. Intensive active exercises are carried out to

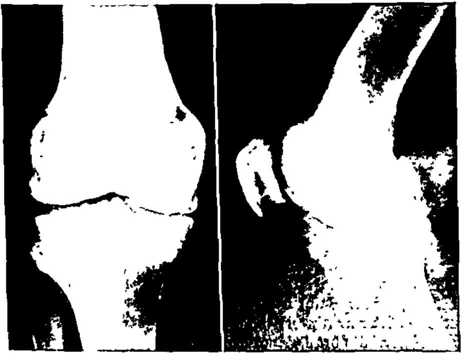

FIG. 241

Advanced osteoarthritis of the knee. Note the narrowing of the cartilage space, sclerosis of the articulating bone surfaces, and osteophytes at the joint margins

strengthen the wasted quadriceps muscle. Local heat therapy is often also given, but it is less important than the exercises. The knee is largely dependent upon the quadriceps for its stability, and if a powerful muscle can be developed there may be no symptoms despite marked osteoarthritis. This is evidenced by the fact that many footballers with osteoarthritis of the knees have been able to continue in first-class football.

In the worst cases, with severe pain and marked flexion deformity, operation is sometimes justified. Arthroplasty of the knee, though theoretically attractive, is seldom successful in practice, and the only reliable operation is arthrodesis, the knee being fused in about 20 degrees of flexion.

Pathology. The articular cartilage is worn away and the underlying bone becomes eburnated. There is hypertrophy of bone at the joint margins, with the formation of osteophytes. The changes may affect predominantly the femoro-tibial joint or the patello-femoral joint; but usually the whole joint is affected.

Clinical features. The patient is commonly an elderly fat woman. Often both knees are affected. There is slowly increasing pain in the joint, worse after unusual activity. The symptoms are often exacerbated by a slight strain or twist. There is usually evidence of one of the predisposing factors mentioned above. *On examination* the knee is slightly thickened from hypertrophy of bone at the joint margins, where a rim of osteophytes may be palpable. Effusion of fluid into the joint is a common but not a constant feature. Movement is moderately restricted and is accompanied by coarse crepitation. The quadriceps muscle is wasted. In severe cases there is a tendency to fixed flexion deformity. *Radiographic examination:* Narrowing of the cartilage space, which is the first sign of osteoarthritis in most joints, is often not discernible until a later stage in the case of the knee. The first clear sign of osteoarthritis in the knee is sharpening or "spiking" of the joint margins, especially of the patella (Fig. 240) and tibia. Later, narrowing of the cartilage space is obvious, osteophytes form at the joint margins, and the subchondral bone may become sclerotic (Fig. 241). "Loose" bodies are often seen. (Most are not in fact loose, but are attached to the synovial membrane.)

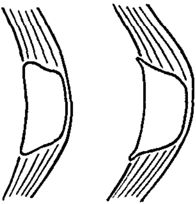

FIG. 240

Spiking of the articular margins of the patella, as seen in the lateral radiograph, is an early sign of osteoarthritis. The normal is shown for comparison.

Diagnosis. This presents little difficulty. But care should be taken not to overlook a coexisting abnormality. Osteoarthritis of the knee may be symptomless, and its demonstration in radiographs does not necessarily mean that it is the cause of existing symptoms.

CHARCOT'S OSTEOARTHROPATHY OF THE KNEE

(General description of Charcot's osteoarthropathy, p. 47.)

The knee is the joint most commonly affected by Charcot's osteoarthropathy (neuropathic arthritis). The commonest underlying cause is tabes dorsalis.

Pathology. In the normal joint harmful strains are prevented by a protective reflex in which muscle contraction is invoked by incipient pain. When joint sensibility is destroyed the protective function of pain is lost. Strains are unrecognised and, cumulatively, they lead to severe degeneration of the joint. The changes may be regarded as a much exaggerated form of osteoarthritis. The articular cartilage and parts of the underlying bone are worn away, but at the same time there is often considerable hypertrophy of bone at the joint margins. The ligaments become lax and the joint is unstable.

FIG. 242

Charcot's osteoarthropathy of the knee. Degenerative destruction is accompanied by massive new bone formation.

Clinical features. The joint is insensitive, so pain is slight or absent. The main features are swelling, laxity, and instability of the knee. Because of the laxity, lateral bowing at the knee is common. *On examination* the knee is greatly thickened, mostly from irregular hypertrophy of the bone ends. There is slight or moderate restriction of movement, and there is marked lateral laxity. The thigh muscles are wasted. Further examination will reveal evidence of the underlying disease—usually tabes dorsalis. *Radiographs* show marked destructive changes, usually with some new bone formation at the joint margins (Fig. 242).

Treatment. In many cases function is adequate despite the

When loose bodies cause recurrent locking of the joint, they should be removed.

HAEMOPHILIC ARTHRITIS OF THE KNEE

(General description of haemophilic arthritis, p. 46)

This uncommon condition is almost confined to males—usually boys. It affects the knee more often than any other joint.

Pathology. Initially there is simply a haemorrhage into the joint (haemarthrosis). With rest, this is slowly absorbed. But further bleeding usually occurs, and it leads eventually to degenerative changes in the articular cartilage and to fibrous thickening of the synovial membrane.

Clinical features. The patient is usually a known sufferer from haemophilia, or can recall previous bleeding incidents. The findings on examination vary according to the phase and duration of the disease. For several weeks after a haemarthrosis the knee remains swollen, partly from contained blood and partly from synovial thickening caused by interstitial extravasation of blood. The overlying skin is abnormally warm. Joint movements are restricted, and painful if forced. In a quiescent phase between attacks of haemarthrosis there is some thickening of the joint from synovial fibrosis, movements are slightly impaired, and often there is moderate flexion deformity. *Investigations:* The clotting time of the blood is usually increased.

Diagnosis. Because of the synovial thickening, increased warmth of the skin and restriction of knee movements, haemophilic arthritis is easily mistaken for chronic inflammatory arthritis. The history and the mode of onset, and the increased clotting time of the blood, are the important distinguishing features. Biopsy should be avoided because it may cause further bleeding.

Treatment. Recent haemarthrosis is treated by aspiration, firm bandaging, and rest. The knee should be immobilised on a Thomas splint or in a plaster. After four to eight weeks the residual blood is absorbed and cautious activity can be resumed.

The chronic degenerative arthritis that results from multiple repeated haemarthroses may have to be controlled by the permanent use of a protective appliance such as a plastic knee splint or a walking caliper. Operation should be avoided.

with the knee semi-flexed or flexed. It is usually a football injury, but it is also common among men who work in a squatting position, such as coal miners.

Pathology. There are three types of cartilage tear (Figs. 245-247). All begin as a longitudinal split (Fig. 243). If this extends

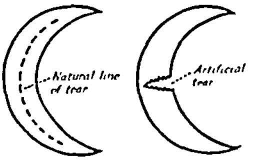

FIG. 243 FIG. 244

Figure 243—Direction of tear in a semilunar cartilage. Figure 244—Transverse tears do not occur in nature, a tear such as this is always an artefact.

throughout the length of the cartilage it becomes a "*bucket-handle*" *tear*, in which the fragments remain attached at both ends (Fig 245). This is much the commonest type. The "bucket handle" (that is, the central fragment) is displaced towards the middle of the joint, so that the condyle of the femur rolls upon the tibia through the rent in the cartilage (Fig. 245). Since the femoral condyle is so shaped that it requires most space when the knee is straight the chief effect of a displaced "bucket handle" is that it limits full extension (="locking").

If the initial longitudinal tear emerges at the concave border of the cartilage a pedunculated tag is formed. In *posterior horn tear* the fragment remains attached at its posterior horn (Fig. 246); in *anterior horn tear* it remains attached at its anterior horn (Fig. 247) A transverse tear through the cartilage is always an artefact, produced by the surgeon at the time of operation (Fig. 244)

The semilunar cartilages are almost avascular: consequently when they are torn there is not an effusion of blood into the joint. But there is an effusion of synovial fluid, secreted in response to the injury Torn cartilages do not heal spontaneously.

marked disorganisation of the knee, and treatment is not required. In other cases marked deformity—usually lateral bowing—may demand some form of protective appliance, such as a moulded plastic splint or a caliper. Arthrodesis of the joint is practicable but fusion is often slow. Because of this and the absence of severe pain operation is seldom advised.

CLUTTON'S JOINTS

The symmetrical swelling of the knee joints in children, described by Clutton, is a manifestation of syphilis. Clinically, there is a large effusion of clear fluid into the joints, but there is little or no pain and good function is preserved. Other signs of syphilis are usually present, and the Wassermann reaction is positive. *Treatment* is by general antisyphilitic remedies and firm bandaging.

CHONDROMALACIA OF THE PATELLA

This is an uncommon affection of adolescents or young adults, in which the cartilage of the articular surface of the patella is roughened and "fibrillated" It is distinct from patello-femoral osteoarthritis, a condition occurring in older patients. Nevertheless chondromalacia predisposes to the later development of osteoarthritis. The cause is unknown.

Clinical features. The knee causes discomfort, especially when flexed and extended under load There is often an effusion of fluid. Movements are accompanied by a fine crepitation transmitted to the examiner's hand upon the patella. *Radiographs* are normal.

Treatment. At first this should be expectant. A firm crepe bandage is applied and strenuous activities are curtailed. These precautions will often allow spontaneous improvement If troublesome symptoms persist for many months, or if radiographs show evidence of developing osteoarthritis, the patella should be excised.

TEARS OF THE SEMILUNAR CARTILAGES

Torn semilunar cartilages are common in young men, especially those who play football. There is always a well remembered causative injury.

Cause. A torn semilunar cartilage is caused by a twisting force

with the knee semi-flexed or flexed. It is usually a football injury, but it is also common among men who work in a squatting position, such as coal miners.

Pathology. There are three types of cartilage tear (Figs. 245-247). All begin as a longitudinal split (Fig. 243). If this extends

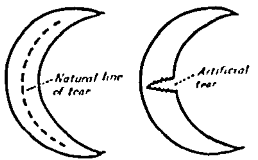

FIG. 243 FIG. 244

Figure 243—Direction of tear in a semilunar cartilage. Figure 244—Transverse tears do not occur in nature, a tear such as this is always an artefact

throughout the length of the cartilage it becomes a "*bucket-handle*" *tear*, in which the fragments remain attached at both ends (Fig. 245). This is much the commonest type. The "bucket handle" (that is, the central fragment) is displaced towards the middle of the joint, so that the condyle of the femur rolls upon the tibia through the rent in the cartilage (Fig 245). Since the femoral condyle is so shaped that it requires most space when the knee is straight the chief effect of a displaced "bucket handle" is that it limits full extension (=" locking ")

If the initial longitudinal tear emerges at the concave border of the cartilage a pedunculated tag is formed. In *posterior horn tear* the fragment remains attached at its posterior horn (Fig. 246); in *anterior horn tear* it remains attached at its anterior horn (Fig. 247). A transverse tear through the cartilage is always an artefact, produced by the surgeon at the time of operation (Fig 244).

The semilunar cartilages are almost avascular: consequently when they are torn there is not an effusion of blood into the joint. But there is an effusion of synovial fluid, secreted in response to the injury. Torn cartilages do not heal spontaneously.

Clinical features of torn medial cartilage. The medial cartilage is torn much more often than the lateral. The patient is 18 to 45 years old. The history is characteristic, especially with "bucket-handle" tears. In consequence of a twisting injury the patient falls and has pain at the antero-medial aspect of the joint. He is unable to continue what he was doing, or does so only with difficulty. He is unable to straighten the knee fully. The next day he notices swelling of the whole knee. He rests the knee. After about two weeks the swelling lessens, the knee seems to go straight, and he resumes his activities. Within weeks or months the knee suddenly "gives way" again during a twisting movement; there are pain and subsequent swelling as before. Similar incidents occur repeatedly. *Locking:* By this is meant inability to extend the knee fully. It is a common feature of torn medial cartilage, but the limitation of extension is often so slight that it is not noticed by the patient. Persistent locking can occur only in "bucket-handle" tears: tag tears cause momentary "catching" but not true locking in the accepted sense. *On examination* in the recent stage the typical features are effusion of fluid, wasting of the quadriceps, local tenderness at the level of the joint antero-medially, and (characteristically in "bucket-handle" tears) limitation of the *last few degrees* of extension by a springy resistance, with sharp antero-medial pain if passive extension is forced.

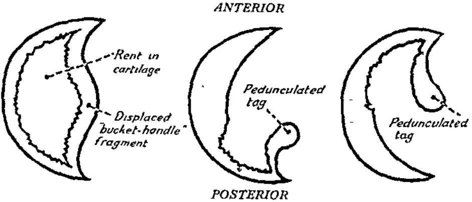

FIG 245 FIG. 246 FIG. 247

The three types of cartilage tear. Figure 245—"Bucket-handle" tear, the commonest type. Figure 246—Posterior horn tear. Figure 247—Anterior horn tear.

In the "silent" phase between attacks there are often no signs other than wasting of the quadriceps.

Clinical features of torn lateral cartilage. The features are broadly similar, but the clinical picture is often less clearly defined. The history may be vague. Pain is at the lateral rather than the medial side of the joint, but it is often poorly localised.

Radiographs are normal, whether the tear be of the medial or lateral cartilage.

Diagnosis. In the "silent" phase diagnosis often depends largely upon the history. The surgeon should be very cautious in diagnosing a torn semilunar cartilage unless there is a clear history of injury and unless there have been recurrent incidents, each followed by synovial effusion. Often a period of observation is required before the diagnosis becomes reasonably certain.

Late effects. Long-continued internal derangement from a torn cartilage predisposes to the later development of osteoarthritis.

Treatment. Once the diagnosis is established the correct treatment is to excise the whole cartilage.

Treatment of the locked knee. Manipulation under anaesthesia is the standard practice, but it seldom replaces the displaced "bucket handle" in its normal position: it merely completes the tear and displaces the "bucket-handle" fragment towards the middle of the joint in the intercondylar region. In this position the cartilage fragment allows greater freedom of joint movement, but completely full extension is seldom restored. Manipulation is therefore worth while only in so far as it increases the patient's comfort while he is awaiting admission for operation.

CYSTS OF THE SEMILUNAR CARTILAGES

A cyst of a semilunar cartilage forms a tense, almost solid swelling at the level of the joint, usually on the lateral side.

Cause. Cysts arise spontaneously, but there is often a previous history of direct injury at the site of the cyst.

Pathology. The swelling is formed by a proliferation of fibrous tissue, which is "honeycombed" with small cystic cavities containing clear gelatinous fluid.

Clinical features. The lateral cartilage is affected much more often than the medial. There is visible swelling, most obvious

when the knee is held slightly flexed, at the level of the joint and usually anterior to the lateral (or medial) ligament (Fig. 248). *The swelling tends to be painful at night and it is usually tender*

FIG. 248
Cyst of lateral cartilage.

on firm pressure The swelling is so tense that fluctuation can seldom be elicited (indeed it is sometimes mistaken for bone). *Radiographs* may show an indentation of the side of the tibial tuberosity where the cyst has been in contact with it.

Treatment. If the disability justifies operation the cyst should be excised together with the cartilage from which it arises.

DISCOID LATERAL SEMILUNAR CARTILAGE

Rarely the lateral semilunar cartilage fails to assume its normal crescentic form during development, but persists in its embryological form as a thick disc-like mass intervening between the lateral condyles of the femur and tibia

A discoid cartilage may cause recurrent discomfort in the knee with a tendency to giving way. Usually a loud " clonk " can be demonstrated on flexion-extension movements. These symptoms often become prominent during adolescence. If the disability becomes troublesome the cartilage should be removed.

OSTEOCHONDRITIS DISSECANS OF THE KNEE

(General description of osteochondritis dissecans, p. 52.)

Osteochondritis dissecans is characterised by local necrosis of a segment of the articular surface of a bone and of the overlying articular cartilage, with eventual separation of the fragment to form an intra-articular loose body. The knee is affected much more often than any other joint.

Cause. This is unknown. Impairment of the blood supply to the affected segment of bone by thrombosis of an end-artery has been suggested. Injury is probably a predisposing factor. But there is also a constitutional predisposition to the disease, for it may affect several joints in the same patient.

Pathology. The lesion nearly always affects the articular surface of the medial condyle of the femur (Figs. 249-251). The size of the affected segment varies—it is often about three-quarters of an

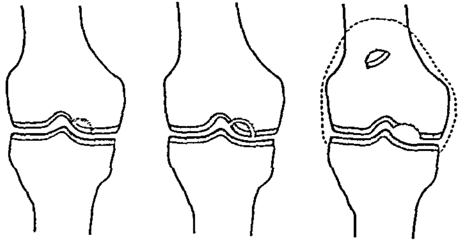

FIG. 249 FIG. 250 FIG. 251

The evolution of osteochondritis dissecans. Figure 249—A segment of the articular surface dies. Figure 250—A line of demarcation forms around it. Figure 251—The fragment breaks away and lies loose in the joint, leaving a cavity in the femoral condyle.

inch in diameter. Within the area of the lesion the subchondral bone is avascular and the overlying cartilage softens. A line of demarcation forms between the avascular segment and the surrounding normal bone and cartilage. After many months the

fragment separates as a loose body (sometimes two or three), leaving a shallow cavity in the articular surface which is ultimately filled with fibro-cartilage. The damage to the joint surface predisposes to the later development of osteoarthritis.

Clinical features. The patient is an adolescent or a young adult. He complains of discomfort or pain in the knee after exercise, a

FIG 252

FIG. 253

'ine antero-posterior radiograph. ojection with knee semiflexed in the medial femoral condyle, set shows the loose fragment after removal

feeling of insecurity, and intermittent swelling. When a loose body has already separated within the joint the predominant symptom is recurrent sudden locking *On examination* there is a fluid effusion. The quadriceps muscle is wasted Movements are not usually impaired *Radiographs* show a clear-cut crescentic excavation of the bone at the articular surface of the medial femoral condyle. At first the cavity is occupied by the separating fragment of bone (Fig. 252); later the cavity may be empty, and a loose body will then be seen elsewhere in the joint The

lesion is shown best in special tangential projections with the knee semiflexed (Fig. 253).

Treatment. Treatment should be expectant at first: the knee is supported with a crepe bandage and strenuous activities are curtailed. When the lesion is "ripe"—that is, when a clear line of demarcation has formed between the separating fragment and the surrounding normal bone—the loose piece should be removed. Inevitably a shallow cavity is left in the femoral condyle; this gradually fills with fibro-cartilage.

LOOSE BODIES IN THE KNEE

The knee is the joint most commonly affected by the formation of loose bodies.[1] There are four main causes: 1) osteochondritis dissecans (1 to 3 loose bodies); 2) osteoarthritis (1 to 10 loose bodies); 3) chip fractures of the joint surfaces (1 to 3 loose bodies); 4) osteochondromatosis (50 to 500 loose bodies).

Pathology. *Osteochondritis dissecans* was described on p. 347. The loose body or bodies are formed by spontaneous separation of a fragment of bone and cartilage from the articular surface of the medial femoral condyle. A shallow cavity remains in the condyle.

Osteoarthritis was described on p. 337. Loose bodies are formed by detachment of marginal osteophytes. Most separated osteophytes, however, retain a synovial attachment and cause no trouble; though they appear "loose" in radiographs they should not be regarded as such unless symptoms of locking indicate that they are moving freely within the joint.

Chip fractures of the joint surfaces are an infrequent cause of intra-articular loose bodies There is a clear history of the causative injury.

Osteochondromatosis is a rare disease of the synovial membrane. It is characterised by the formation of numerous small villous processes, which become pedunculated. Later their bulbous extremities become cartilaginous and they are detached to lie free in the joint. Finally, some or all of the numerous loose bodies become calcified.

[1] A clear distinction must be made between *loose* bodies (*i e.*, fragments of the joint tissues that have separated off to lie free in the joint) and *foreign* bodies (*i e*, pieces of extraneous matter introduced into the body from outside). Students often confuse the two terms.

Whatever the cause of a loose body, its repeated jamming between the joint surfaces will predispose to osteoarthritis or will aggravate existing osteoarthritis.

Clinical features. The characteristic symptom of a loose body in the knee is recurrent locking of the joint from interposition of the loose piece between the joint surfaces. Suddenly, without warning, the knee becomes "jammed" during movement. Locking is accompanied by severe pain. After a variable interval

Fig. 254

Fig. 255

Figure 254—Loose body in the knee. As in this case, loose bodies often lie in the suprapatellar pouch. Figure 255—The fabella, a sesamoid bone in the lateral head of the gastrocnemius, present in many normal persons. It is sometimes mistaken for a loose body in the joint.

the patient is usually able, by manoeuvring the limb, to disengage the loose body and free the joint. The next day the knee is found to be swollen with fluid. In many cases the patient is able to feel the mobile body through the soft tissues when it lies in a superficial part of the joint. *On examination* the findings are often slight, for a patient is seldom seen when the knee is locked. If he is seen soon afterwards, a fluid effusion is present. Between attacks the only constant sign is wasting of the quadriceps. Sometimes the loose body can be palpated. The features of an underlying

condition such as osteoarthritis may be present. *Radiographic examination:* With few exceptions, loose bodies are shown radiographically. They often lie in the suprapatellar pouch (Fig. 254). Care should be taken not to mistake the fabella (a sesamoid bone in the lateral head of the gastrocnemius) (Fig. 255) for a loose body. The position of the fabella is constant—it lies slightly above the joint level well behind the femur and towards the lateral side—and it is always oval in shape with its long axis vertical.

Treatment. In general the treatment for an intra-articular loose body is to remove it. Removal should always be advised if the body is causing recurrent locking. If there are no symptoms of locking operation is not essential. In such cases the bony fragment, though appearing loose in the radiographs, is often attached to the synovial membrane and is kept out of harm's way. This applies particularly to detached osteophytes in cases of osteoarthritis.

RECURRENT DISLOCATION OF THE PATELLA

The patello-femoral joint is one of the three joints that are most liable to recurrent displacement, the others being the shoulder and the ankle. In the case of the patello-femoral joint, unlike the other two, the instability is often caused by congenital factors rather than by an initial violent injury.

Pathological anatomy. In dislocations of the patella the displacement is always lateral, the patella slipping over the lateral condyle of the femur while the knee is flexed. Three factors predispose to recurrent dislocation: 1) under-development of the lateral femoral condyle, with a shallow intercondylar groove; 2) an abnormally high position of the patella, which consequently does not lie so deeply as usual in the intercondylar groove; and 3) genu valgum, which causes the line of pull of the quadriceps to lie too far to the lateral side.

Clinical features. Recurrent dislocation of the patella is more common in girls than in boys. Often both knees are affected. Trouble usually begins during adolescence or in early adult life. The dislocations occur while the patient is engaged in some activity that entails flexing the knees—not necessarily a violent exertion. Suddenly, while the knee is flexed or semi-flexed, there is severe

pain in the front of the knee, and the patient is unable to straighten it. In many cases the displacement of the patella is recognised and reduced on the spot, either by the patient herself or by an onlooker.

On examination in the dislocated state the knee is swollen, and the patella is seen and felt upon the lateral side of the lateral femoral condyle. After reduction the main signs are an effusion (usually of blood-stained fluid), and tenderness over the medial part of the quadriceps expansion, which is usually strained or torn. Genu valgum, or one of the other minor variations mentioned above, may be observed. A further common feature is an ability to hyperextend the knee well beyond the straight position (genu recurvatum).

Radiographs are seldom obtained in the state of dislocation. After reduction the knee may appear normal radiographically, but commonly the patella is seen at a slightly higher level than usual—often in both knees.

Course. Dislocation of the patella does not always become recurrent: some patients have no further trouble after one or two dislocations But in many cases dislocations recur with ever-increasing ease and frequency, so that the patient may become seriously handicapped. Oft-repeated dislocations predispose to the later development of osteoarthritis.

Treatment. Treatment should be expectant at first. After a dislocation the patient should receive a course of physiotherapy designed to strengthen the quadriceps muscle, and especially the vastus medialis

If the dislocations recur with such frequency that the patient is seriously disabled, operation should be advised. The method recommended is to detach the bony insertion of the patellar tendon and transpose it to a new bed in the tibia, medial and distal to the original insertion. In this way the patella is drawn lower into the intercondylar groove of the femur, and the line of pull of the quadriceps mechanism is transferred more to the medial side.

An alternative operation is to excise the patella. It has been claimed that this gives better long-term results because the liability to osteoarthritis is reduced, but the claim is not yet proved.

EXTRA-ARTICULAR DISORDERS IN THE REGION OF THE KNEE

RUPTURE OF THE QUADRICEPS APPARATUS

The quadriceps muscle gains insertion into the tibia through the medium of the patella (enclosed within the quadriceps expansion) and the patellar tendon. Complete rupture may occur at three points (Fig. 256): 1) at the point of attachment of the quadriceps tendon to the upper pole of the patella; 2) through the patella and the surrounding quadriceps expansion; or 3) at the attachment of the patellar tendon to the tibial tubercle. In all cases the injury is caused by an unexpected flexion force, resisted automatically by a sudden contraction of the quadriceps.

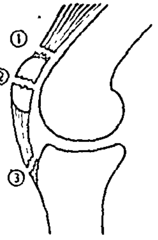

FIG 256

The three points at which the quadriceps apparatus may rupture. 1. At insertion of quadriceps into patella. 2. Through the patella 3 At insertion of patellar tendon into tibial tubercle.

AVULSION FROM PATELLA

This occurs mainly in elderly men in whom the quadriceps tendon is often degenerate. The tendon should be re-attached to the bone by sutures of stainless steel wire.

DISRUPTION THROUGH PATELLA

This is the usual site of rupture, and it forms a common variety of fractured patella. The injury occurs mainly in adults of middle age. If the patella is cleanly broken into two pieces and the patient is under 45 the fragments should be fixed together by a screw. Otherwise the patellar fragments should be excised and the quadriceps expansion sutured.

AVULSION AT TIBIAL TUBERCLE

This is the least common injury, occurring mainly in children or young adults The torn tendon should be reattached by sutures.

INFRAPATELLAR BURSITIS

The bursa that lies in front of the lower pole of the patella and the upper part of the patellar tendon is prone to inflammation.
Types. There are two types of infrapatellar bursitis: 1) irritative; and 2) infective or suppurative.

Irritative Infrapatellar Bursitis

This is caused by repeated friction; it occurs especially in those who do much kneeling. There is fibrous thickening of the wall of the bursa, which is distended with fluid.
Clinical features. There is a softly fluctuant swelling in front of the lower part of the patella and upper part of the patellar tendon. The swelling is clearly demarcated. It is manifestly confined to a plane in front of the joint, and the joint itself is unaffected.
Treatment. *Repeated aspiration of the fluid may be sufficient* treatment if further friction can be avoided. Operative excision of the bursa affords a more certain permanent cure.

Suppurative Infrapatellar Bursitis

This is caused by infection of the bursa with pyogenic organisms, which reach the bursa directly through a puncture wound, or through the lymphatics from an infected lesion on the leg. The wall of the bursa is acutely inflamed and the sac is distended with pus
Clinical features. There are pain and swelling in front of the knee, often with pyrexia. The swelling is confined to the site of the infrapatellar bursa. It is acutely tender on palpation, and the overlying skin is hot and reddened. The inguinal lymphatic glands are often enlarged and tender. The knee joint itself is unaffected, but the patient is unwilling to bend it fully because flexion increases the pain by tensing the skin over the bursa
Treatment. Appropriate chemotherapy should be instituted and the bursal abscess should be drained by incision.

BAKER'S CYST

A Baker's cyst is simply a herniation of the synovial cavity of the knee, with the formation of a fluid-filled sac extending backwards and downwards (Fig 257). It is not a primary condition

but is always secondary to a disorder of the knee with persistent synovial effusion, such as osteoarthritis. In long-standing cases the hernial sac is much elongated, and may extend a considerable

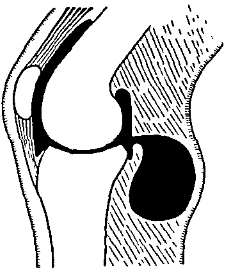

FIG. 257
To show how a Baker's cyst is formed as a herniation of the synovial membrane.

distance down the calf. *Clinically* there is a soft cystic bulge in the midline behind the knee or in the upper calf. The underlying abnormality of the knee, with synovial effusion, will usually be obvious.

Treatment. In most cases treatment should be directed towards the underlying condition of the knee rather than to the cyst itself. Nevertheless if the cyst is extensive it is sometimes advisable to excise it.

SEMIMEMBRANOSUS BURSITIS

Inflammation of the bursa between the medial head of the gastrocnemius and the semimembranosus is an occasional cause of swelling behind the knee. The inflammation is caused by friction, not by bacterial infection. The bursa enlarges to form an elongated sac bulging between the muscle planes. *Clinically* there is a soft cystic swelling at the back of the knee, close to the medial condyle of the femur.

Treatment is by excision of the sac.

PELLEGRINI-STIEDA'S DISEASE

Pellegrini-Stieda's disease is the name sometimes used to describe ossification in the subligamentous haematoma after partial avulsion of the medial ligament from the medial condyle of the femur. *Clinically* there is persistent discomfort at the medial side of the knee after an injury to the medial ligament. There are thickening and slight tenderness over the site of attachment of the ligament to the medial femoral condyle. *Radiographs* show a thin plaque of new bone close to the medial condyle (Fig. 258).

Treatment is by active mobilising and muscle-strengthening exercises.

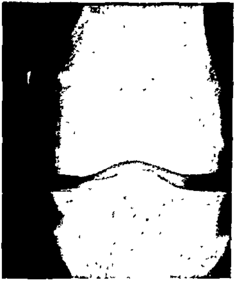

FIG. 258

FIG. 258

Pellegrini-Stieda's disease There is an ossified plaque at the femoral attachment of the medial ligament.

OSGOOD-SCHLATTER'S DISEASE

(Osteochondritis of the tibial tubercle)

Osgood-Schlatter's disease is a childhood affection of the tibial tubercle, which becomes enlarged and temporarily painful. The precise nature of the condition is unknown. It has been widely accepted as an example of osteochondritis (p. 77), and for want of more accurate knowledge it will be so regarded here. Many surgeons believe, however, that Osgood-Schlatter's disease is nothing more than a strain of the developing tibial tubercle, from the pull of the patellar tendon

Pathology. The main pathological feature is a moderate enlargement of the tibial tubercle.

Clinical features. The patient is a child of 10 to 14 years The complaint is of pain in front of and below the knee, worse on

strenuous activity. *On examination* the tibial tubercle is unduly prominent, and tender on palpation. Pain is increased when the quadriceps is tensed, as in straight leg raising against resistance. The symptoms and signs are confined to the region of the tibial tubercle and the knee joint itself is normal. *Radiographs* show enlargement and fragmentation of the tibial tubercle, sometimes with patchy increase of density of the bone.

Course. The disorder is self-limiting, and normal function is always restored. Since the joint itself is unaffected there is no fear of the development of secondary osteoarthritic changes, as there is in some other examples of osteochondritis, such as Perthes' disease.

Treatment. In many cases treatment is not required. If local pain and tenderness are severe the knee should be rested for two months in a plaster cylinder extending from groin to malleoli.

EXTRINSIC DISORDERS WITH REFERRED SYMPTOMS IN THE KNEE

From time to time patients are seen whose main complaint is of pain in the knee, but local examination reveals no satisfactory explanation for it In such cases the possibility that the pain is referred from a disorder distant from the knee should always be considered. The commonest source of such referred pain is a disorder of the hip. Much less often a disorder of the spine is responsible.

DISORDERS OF THE HIP

The conditions of the hip in which the pain may be felt predominantly in the knee are the various types of arthritis, of which osteoarthritis is the most common. The pain is referred along the course of one of the peripheral nerves—especially the obturator nerve, which takes a large share in the innervation of the hip.

Differential diagnosis. There should be little difficulty in determining the true source of the pain if the possibility of a hip lesion is borne in mind. Whenever knee symptoms cannot be adequately explained after a local examination of the knee the hip should be investigated and, if necessary, radiographed.

PELLEGRINI-STIEDA'S DISEASE

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Treatment is by active mobilising and muscle-strengthening exercises

FIG. 258

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CHAPTER TEN

The Leg, Ankle, and Foot

IN the orthopaedic out-patient clinic disorders of the foot are second in frequency only to disorders of the back. Their prevalence may have several causes. *Hereditary factors :* The foot is probably in a state of relatively rapid evolution consequent upon man's assumption of the upright posture, and perhaps for that reason it is prone to variations in structure and form which may impair its efficiency. *Postural stresses :* Overweight throws an increased burden on the feet, and they may be unable to withstand the stress without ill-effect, especially if the intrinsic muscles are poorly developed. *Footwear :* The wearing of shoes is a potent cause of foot disorders. Many types of shoe interfere seriously with the mechanics of the foot, and the fashionable ladies' shoe with high heel and narrow pointed toe is particularly to blame.

SPECIAL POINTS IN THE INVESTIGATION OF LEG, ANKLE, AND FOOT COMPLAINTS

In nearly all cases symptoms in the leg, ankle, or foot can be explained by a local abnormality. Only rarely are they referred from a distant lesion. In this respect the lower limb differs markedly from the upper, for in many cases symptoms in the hand have no local cause but are referred from a proximal lesion.

History

The precise distribution of pain should be ascertained. The occupation and habits of the patient, or a history of previous injury, may be significant. Specific enquiry should be made into the effects upon the symptoms of standing and walking.

Exposure

It is essential that socks or stockings be removed and that the whole leg be exposed up to the knee. Both limbs must always be examined so that the two may be compared. The first part of the examination

DISORDERS OF THE SPINE

The only disorders of the spine that may cause symptoms predominantly in the region of the knee are those that cause pressure on the roots of the lumbar or sacral plexus, especially prolapsed intervertebral disc. It is only rarely that such referred symptoms are unassociated with other symptoms in the back, buttock, or thigh.

Ankle movement. The ankle is strictly a hinge joint. The only movements are extension (dorsiflexion) and plantar-flexion. The range should be judged from the excursion of the hindfoot rather than the

TABLE XI

ROUTINE CLINICAL EXAMINATION IN SUSPECTED DISORDERS OF THE LEG, ANKLE, AND FOOT

1 LOCAL EXAMINATION OF THE LEG, ANKLE, AND FOOT

Inspection

Bone contours and alignment
Soft-tissue contours
Colour and texture of skin
Scars or sinuses

Palpation

Skin temperature
Bone contours
Soft-tissue contours
Local tenderness

State of peripheral circulation

Dorsalis pedis pulse
Posterior tibial pulse
Popliteal pulse
Femoral pulse
? Cyanosis of foot when dependent

Movements (active and passive, compared against normal side)

At the ankle .
Plantar-flexion
Extension (dorsiflexion)

At the subtalar joint
Inversion-adduction
Eversion-abduction

At the midtarsal joint
Inversion-adduction
Eversion-abduction

At the toes :
Flexion
Extension

Power (tested against resistance of examiner)

Each muscle group to be tested in turn. (*N.B.*—Power of calf muscles is best tested with the patient standing)

Stability

Integrity of ligaments—particularly the lateral ligament of the ankle

Appearance of foot on standing

[illegible]

to raise heel from ground while standing on affected leg)

Gait

Condition of footwear

Sites of greatest wear

2. GENERAL EXAMINATION

General survey of other parts of the body. The local symptoms may be only one manifestation of a widespread disease

forefoot, so that any contribution from the tarsal joints is disregarded. Similarly, in testing the passive range, the foot should be controlled from the heel (Fig. 259). The normal range of ankle movement varies

is conducted with the patient sitting, the heel being supported on a stool; or, better still, the patient may recline on a couch. Later, the foot is examined while the patient stands.

Steps in Clinical Examination

A suggested plan for the routine clinical examination of the leg, ankle, and foot is summarised in Table XI.

Assessing the State of the Peripheral Circulation

An essential part of the examination of the foot that is often forgotten is to study the efficiency of the arterial circulation. This may be judged from the colour, temperature, and texture of the skin, from palpation of the arterial pulses, and from certain special tests. *Colour:* A brick-red rubor or cyanosis when the foot is dependent, with rapid blanching on elevation, denotes serious impairment of the arterial circulation. *Temperature:* A foot with impaired arterial supply is colder than normal, but little reliance can be placed on this test as applied clinically at the bedside. *Texture of skin and nails:* The skin of an ischaemic foot loses its hair and becomes thin and inelastic. The nails are course, thickened, and irregular *Arterial pulses:* The pulses to be felt for are the dorsalis pedis, the posterior tibial, and, if those are absent, the popliteal and femoral pulses. Pulsation of the dorsalis pedis artery is best felt at the dorsum of the foot between the bases of the first and second metatarsals. The posterior tibial artery is felt about three-quarters of an inch behind and below the tip of the medial malleolus. Absence of arterial pulsation is a most important sign of impaired peripheral circulation. It should be remembered, however, that a normal pulse is easily masked by thickening or oedema of the soft tissues.

When these clinical tests throw doubt upon the integrity of the arterial supply further investigations are required. *Temperature recordings:* Accurate measurement of the skin temperature of the foot before and after inhibition or temporary paralysis of the sympathetic fibres (by rest-of-body heating, procaine nerve block, or spinal anaesthetic) gives a reliable indication of the state of the arteries. With healthy vessels the rise of temperature from vasodilation should be about 7 to 10 degrees, whereas in severe arterial disease the rise may be less than one degree. *Exercise tolerance tests:* Repeated movements of the foot are carried out against resistance to test whether symptoms of claudication arise *Arteriography*. The arterial tree is outlined by radiography after the injection of an opaque fluid (diodone) into the main vessel.

Movements at the Ankle and Tarsal Joints

Since the joints are close together, movement at the tarsal joints is easily mistaken for movement at the ankle, and vice versa. Careful examination is required to determine the range at each individual joint.

remembered that the normal range of dorsiflexion of the great toe at the metatarso-phalangeal joint is nearly 90 degrees (Fig. 262). Limitation to less than 60 degrees of dorsiflexion is certainly abnormal.

Examination of the Feet under Weight-bearing Stress

Instruct the patient to stand evenly on both feet. Observe the general shape of the ankle, foot, and toes. Study the shape of the longitudinal arch. Is it of normal shape? Is it flattened so that the navicular region is in contact with the ground (pes planus or valgus)? Or is it higher than normal (pes cavus)? Next study the forefoot. Is it splayed and broader than normal? Assess the function of the toes. Normally they can be pressed upon the ground by the action of the intrinsic muscles so that the metatarsal heads are lifted up and relieved of weight-bearing pressure. Finally, examine the efficiency of the calf muscles. The crucial test is to ask the patient to stand on the affected leg and to raise the heel from the ground.

Gait

Look especially for abnormal posture of the feet such as turning in (intoeing) or turning out, or drop foot. Observe whether weight is borne correctly on the sole of the foot or whether it is taken too much on the medial or lateral border. Observe also whether the heel is raised normally from the ground at the beginning of each step.

Footwear

The examination of the foot is not complete until the patient's shoes have been inspected and compared on the two sides. Note the position of greatest wear. When the foot is normal the greatest wear in the sole occurs beneath the "ball" of the foot and slightly to the medial side. In the heel it is at the posterior border slightly to the lateral side. The state of the uppers is also important: excessive bulging on the medial side suggests a valgus foot and excessive bulging on the lateral side an inverted foot.

Radiographic Examination

The ankle. Routine radiographs of the ankle comprise an antero-posterior and a lateral projection centred at the level of the joint. The films should include a reasonable length of the shafts of the tibia and fibula, and the whole of the talus.

When laxity of the lateral ligament is suspected a special "inversion-stress" film is required. This is an antero-posterior projection taken while the heel is held in fullest inversion by an assistant. If the lateral ligament is torn or lax the talus will be shown tilted in the ankle mortice (p. 377)

The foot. Routine radiographs of the foot comprise an antero-posterior (strictly a supero-inferior) projection and a lateral projection. Special techniques are used to show the calcaneum (axial view) and the subtalar joint (oblique projection).

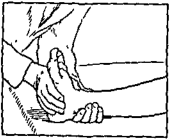

Fig. 259
Examining ankle movement

in different subjects, so the normal ankle must be used as a control. An average range is about 25 degrees of extension and 35 degrees of plantar-flexion.

Subtalar and midtarsal movement. In normal use the subtalar and midtarsal joints work together as a single unit. The movements permitted are: 1) combined inversion and adduction, and 2) combined eversion and abduction. In clinical examination the range of movement contributed by each component can be determined separately. To test *subtalar movement* support the lower leg by a hand gripping the ankle. With the other hand lightly grasp the calcaneum from below (Fig. 260). Instruct the patient

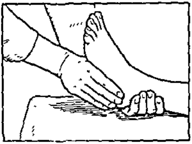

Fig. 260
Examining subtalar movement.

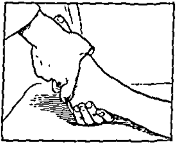

Fig. 261
Examining midtarsal movement

alternately to invert and evert the foot, observing the range through which the heel rocks from side to side. Compare with the sound foot. The normal range is about 20 degrees on each side of the vertical.

To test *midtarsal movement* grasp the calcaneum firmly so that subtalar movement is eliminated. With the other hand lightly grasp the midfoot near the bases of the metatarsals (Fig. 261). Instruct the patient alternately to twist the foot inwards and outwards into inversion and eversion, and compare the range with that on the sound side. The normal is a rotation of about 20 degrees on each side of the neutral.

Toe movements. Determine the active and passive range at the metatarso-phalangeal and interphalangeal joints. It should be

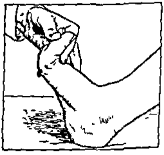

Fig. 262
The normal range of dorsiflexion at the metatarso-phalangeal joint of the great toe is nearly 90 degrees.

Fig. 262

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CLASSIFICATION OF DISORDERS OF THE LEG, ANKLE, AND FOOT

DISORDERS OF THE LEG

INJURIES

Rupture of the tendo calcaneus

INFECTIONS

Acute osteomyelitis
Chronic osteomyelitis
Syphilitic infection

TUMOURS

Benign tumours of bone
Malignant tumours of bone

OTHER DISEASES OF BONE

Paget's disease of the tibia

CIRCULATORY DISORDERS

Intermittent claudication

DISORDERS OF THE ANKLE

ARTHRITIS

Pyogenic arthritis
Rheumatoid arthritis
Tuberculous arthritis
Osteoarthritis
Gouty arthritis
Haemophilic arthritis
Charcot's osteoarthropathy

POST-TRAUMATIC MECHANICAL DERANGEMENTS

Recurrent subluxation

DISORDERS OF THE FOOT

DEFORMITIES

Congenital club foot (talipes equino-varus)
Talipes calcaneo-valgus
Accessory bones in the foot

Pes cavus
Pes planus (flat foot)

POSTURAL DISORDERS

Foot strain

ARTHRITIS

Osteoarthritis of the tarsal joints
Other forms of tarsal arthritis

OSTEOCHONDRITIS

Osteochondritis of the navicular bone
Osteochondritis of the calcaneum
(included under painful heel)
Osteochondritis of a metatarsal head
(included with disorders of the toes)

MISCELLANEOUS

Painful heel
Pain in the forefoot
Plantar wart
Callosities
Ganglion

DISORDERS OF THE TOES

DEFORMITIES

Hallux valgus
Hammer toe
Under-riding toe

ARTHRITIS

Pyogenic arthritis
Osteoarthritis
Gouty arthritis

OSTEOCHONDRITIS

Osteochondritis of a metatarsal head

MISCELLANEOUS

Ingrowing toe nail
Subungual exostosis
Onychogryposis

DISORDERS OF THE LEG

RUPTURE OF THE TENDO CALCANEUS

Surprising as it may seem, a ruptured tendo calcaneus (tendo Achillis) is often overlooked, the symptoms being wrongly ascribed to a " strained muscle " or a " ruptured plantaris."

Pathology. The rupture is always complete. It occurs about two inches above the insertion of the tendon. If it is left untreated the tendon unites spontaneously, but with lengthening

Clinical features. While running or jumping the patient feels a sudden agonising pain at the back of the ankle. He may believe that something has struck him. He is able to walk, but with a limp. *On examination* there is tenderness at the site of rupture. There is general thickening from effusion of blood and from oedema of the paratenon, but a gap can usually be felt in the course of the tendon. The power of plantar-flexion at the ankle is greatly weakened, though some power remains through the action of the tibialis posterior, the peronei, and the toe flexors.

FIG. 263

The crucial test of intact calf function is to raise the heel from the ground while standing only on the affected leg Inability to do this after an injury to the calcaneal tendon is diagnostic of complete rupture

Diagnosis. The retention of some power of plantar-flexion may deflect the unwary from the correct diagnosis. The crucial test is to ask the patient to lift the heel from the ground while standing only upon the affected leg (Fig. 263) This is impossible if the tendon is ruptured.

Treatment. The tendon should be sutured, tension being relaxed by immobilising the limb with right-angled knee flexion and full ankle plantar-flexion for three weeks. Thereafter a below-knee plaster with the ankle at 90 degrees is worn for a further five weeks

ACUTE OSTEOMYELITIS

(General description of acute osteomyelitis, p. 54.)

The tibia is one of the commonest sites both of the haematogenous type of infection and of direct infection introduced from without. The fibula is less often affected.

Pathology. Haematogenous (blood-borne) infection begins either in the upper or in the lower metaphysis, usually of the tibia but sometimes of the fibula. Spread of infection to the knee or ankle is uncommon, because both the upper and lower metaphyses of the tiba are entirely outside the capsule of the knee and ankle respectively (Fig. 31, p. 56).

Direct infection of the bone from without is nearly always a complication of a compound fracture. It is more common in the tibia than in any other bone because the tibia is so often the site of a compound fracture. Any part of the shaft may be affected, depending on the level of the fracture.

Clinical features. Acute haematogenous osteomyelitis occurs mostly in children. There is a rapid onset of severe local pain, with constitutional illness and pyrexia. *On examination* the child often shows signs of general illness. There is marked but circumscribed local tenderness over the affected bony metaphysis. There is usually some induration of the soft tissues over the site of infection. Later the skin may become reddened and an abscess may be palpable. Movements of the adjacent joint are but little impaired. As in all cases of acute haematogenous osteomyelitis, *radiographs* show no abnormality in the initial stages.

Osteomyelitis secondary to a compound fracture is more common in adults. The temperature fails to settle after the primary treatment of the fracture, and re-examination reveals a collection of pus beneath the skin or a purulent discharge from the wound.

Treatment. The treatment of acute haematogenous osteomyelitis was described on page 58. In cases seen early, in which there is no sign of an abscess outside the bone, reliance is placed upon rest and antibiotic drugs; but if an abscess has formed these measures must be combined with local drainage.

The treatment of osteomyelitis complicating open fractures consists mainly in securing adequate drainage and immobilisation, with later removal of sequestra should they form.

DISORDERS OF THE LEG

RUPTURE OF THE TENDO CALCANEUS

Surprising as it may seem, a ruptured tendo calcaneus (tendo Achillis) is often overlooked, the symptoms being wrongly ascribed to a " strained muscle " or a " ruptured plantaris "

Pathology. The *rupture is always complete It occurs about* two inches above the insertion of the tendon. If it is left untreated the tendon unites spontaneously, but with lengthening

Clinical features. While running or jumping the patient feels a sudden agonising pain at the back of the ankle. He may believe that something has struck him. He is able to walk, but with a limp. *On examination* there is tenderness at the site of rupture. There is general thickening from effusion of blood and from oedema of the paratenon, but a gap can usually be felt in the course of the tendon The power of plantar-flexion at the ankle is greatly weakened, though some power remains through the action of the tibialis posterior, the peronei, and the toe flexors.

Fig 263

The crucial test of intact calf function is to raise the heel from the ground while standing only on the affected leg Inability to do this after an injury to the calcaneal tendon is diagnostic of complete rupture

Diagnosis. The retention of some power of plantar-flexion may deflect the unwary from the correct diagnosis. The crucial test is to ask the patient to lift the heel from the ground while standing only upon the affected leg (Fig. 263). This is impossible if the tendon is ruptured.

Treatment. The tendon should be sutured, tension being relaxed by immobilising the limb with right-angled knee flexion and full ankle plantar-flexion for three weeks Thereafter a below-knee plaster with the ankle at 90 degrees is worn for a further five weeks

ACUTE OSTEOMYELITIS

(General description of acute osteomyelitis, p. 54.)

The tibia is one of the commonest sites both of the haematogenous type of infection and of direct infection introduced from without. The fibula is less often affected.

Pathology. Haematogenous (blood-borne) infection begins either in the upper or in the lower metaphysis, usually of the tibia but sometimes of the fibula. Spread of infection to the knee or ankle is uncommon, because both the upper and lower metaphyses of the tiba are entirely outside the capsule of the knee and ankle respectively (Fig. 31, p. 56).

Direct infection of the bone from without is nearly always a complication of a compound fracture. It is more common in the tibia than in any other bone because the tibia is so often the site of a compound fracture. Any part of the shaft may be affected, depending on the level of the fracture.

Clinical features. Acute haematogenous osteomyelitis occurs mostly in children. There is a rapid onset of severe local pain, with constitutional illness and pyrexia. *On examination* the child often shows signs of general illness. There is marked but circumscribed local tenderness over the affected bony metaphysis. There is usually some induration of the soft tissues over the site of infection. Later the skin may become reddened and an abscess may be palpable. Movements of the adjacent joint are but little impaired. As in all cases of acute haematogenous osteomyelitis, *radiographs* show no abnormality in the initial stages.

Osteomyelitis secondary to a compound fracture is more common in adults. The temperature fails to settle after the primary treatment of the fracture, and re-examination reveals a collection of pus beneath the skin or a purulent discharge from the wound.

Treatment. The treatment of acute haematogenous osteomyelitis was described on page 58. In cases seen early, in which there is no sign of an abscess outside the bone, reliance is placed upon rest and antibiotic drugs; but if an abscess has formed these measures must be combined with local drainage.

The treatment of osteomyelitis complicating open fractures consists mainly in securing adequate drainage and immobilisation, with later removal of sequestra should they form.

CHRONIC OSTEOMYELITIS

(General description of chronic osteomyelitis, p. 59)

Chronic osteomyelitis in the lower leg, as elsewhere, is nearly always a sequel of acute osteomyelitis. It may follow either the haematogenous type of infection or an infected open fracture. It has the features of chronic osteomyelitis elsewhere and the principles of treatment are the same.

Brodie's Abscess

This rather uncommon lesion was described on page 60. It is a special form of chronic osteomyelitis which arises insidiously, without a recognised acute infection preceding it. The tibia is the commonest site. Clinically there is a deep "boring" pain, often occurring in attacks with intervals of relative freedom. There may be local tenderness over the site of the lesion, with some induration of the soft tissues. Radiographs show a cavity surrounded by a zone of sclerosis (Fig. 38, p. 60) Treatment is by drainage: the cavity is de-roofed and, whenever possible, it should be obliterated by allowing soft tissues to fall within it.

SYPHILITIC INFECTION OF THE TIBIA

(General description of syphilitic osteitis, p. 64.)

Although skeletal syphilis is now rare in Great Britain, when it does occur the tibia is often the bone affected. The infection may take the form of a localised gumma or a diffuse osteoperiostitis (Fig. 45) There is a gradually enlarging swelling, with moderate pain. It is important to bear the possibility of syphilis in mind, for the swelling is easily mistaken for a tumour These syphilitic manifestations in bone respond rapidly to antisyphilitic treatment —an important diagnostic feature.

TUMOURS OF BONE

BENIGN TUMOURS

(General description of benign bone tumours, p. 66.)

Of the four main types of benign tumours of bone—osteoma, chondroma, osteochondroma, and osteoclastoma—only the chondroma and osteoclastoma require further mention here.

CHONDROMA

In the tibia or fibula this is seldom found except in multiple form in the condition of dyschondroplasia (p. 84). The individual tumours in this condition resemble enchondromata. They arise from the growing epiphysial cartilage plate, and they interfere with the normal growth of the bone. An important effect is that the growth of the tibia and fibula may be unequal, with the consequence that the plane of the ankle joint may be tilted away from the horizontal (Fig. 14).

OSTEOCLASTOMA

The upper end of the tibia or fibula, like the lower end of the femur, is a favourite site for this tumour. It usually arises in a young adult, expanding the bone and extending to within a short distance of the articular surface.

Treatment. If the tumour is in the fibula the whole of the affected end of the bone should be excised together with an adequate margin of healthy bone. If the tumour is in the upper end of the tibia, reliance should usually be placed upon careful curettage and packing of the cavity with bone chips. But if the innocence of the tumour is in doubt wide local excision, with pre-operative and post-operative radiotherapy, should be advised.

MALIGNANT TUMOURS

(General description of malignant bone tumours, p. 70.)

The tibia is a favourite site of primary malignant bone tumours, but secondary tumours are less common than they are in the femur.

OSTEOGENIC SARCOMA

This tumour arises in the metaphysis of a long bone, usually in a child or young adult. When it arises in later life if is often a complication of Paget's disease. The upper metaphysis of the tibia is one of its commonest sites: the lower end of the tibia and the fibula are affected much less often. The tumour metastasises rapidly through the blood stream, especially to the lungs.

Treatment. Amputation is the usual method, but it should not be undertaken if pulmonary metastases are already demonstrable. Wide local excision is sometimes practicable. Surgical treatment should always be supplemented by radiotherapy. In hopeless cases radiotherapy alone is useful in controlling the primary tumour and preventing fungation.

EWING'S TUMOUR

This is also a tumour of childhood. It affects the shaft of a long bone. The tibia is one of the commonest sites. There is a fusiform thickening of the bone, usually with increased warmth of the overlying skin. *Radiographs* show the typical " onion-peel " appearance due to the formation of new bone in concentric layers. **Treatment.** *Although it is ultimately fatal from metastases,* Ewing's tumour is radio-sensitive, and treatment by deep x-ray therapy is probably as effective as amputation.

MULTIPLE MYELOMA

The multiple lesions of this fatal disease are infrequent in the lower leg, where there is little red bone marrow.

SECONDARY (METASTATIC) TUMOURS

Secondary tumours are uncommon in the bones of the lower leg.

PAGET'S DISEASE OF THE TIBIA

Paget's disease (osteitis deformans) was described in the section on general affections of the skeleton on page 89. After the pelvis and spine, the tibia is one of the commonest sites of the disease, and for a time it may be the only bone affected. Characteristically the tibia is thickened and bowed forwards and laterally. *Clinically* there may be no symptoms, but in most cases there is obvious bowing, and sometimes there is a deep aching or " boring " pain. Brittleness of the bone leads occasionally to pathological fracture *Radiographs* show thickening of the cortex and coarsening of the trabeculae; the contrast between cortex and medulla is much less distinct than it is in normal bone (Fig. 264). No effective treatment is available.

FIG. 264

FIG. 264

Paget's disease of the tibia Note the bowing, the coarse trabeculae, and the loss of clear distinction between cortex and medulla.

INTERMITTENT CLAUDICATION

Intermittent claudication is a symptom of arterial insufficiency in the lower limb. In its typical form it is characterised by cramp-like pain in the calf, induced by walking.

Cause. The usual underlying cause is arteriosclerosis with local thrombosis of the main limb vessel. Thrombo-angiitis obliterans and arterial embolism are less common causes.

Pathology. The basic disturbance is ischaemia of muscle, in consequence of which metabolites cannot be removed speedily enough when the muscle is exercised. The accumulation of metabolites is believed to be responsible for the pain, which subsides when the muscle is rested. The muscles usually affected are those of the calf, but in some instances other muscle groups are involved. The vascular lesion is usually a complete occlusion of the femoral or the popliteal artery.

Clinical features. Intermittent claudication is much more common in men than in women. In the usual arteriosclerotic type the patient is past middle life, but in cases due to thrombo-angiitis obliterans or embolism the symptoms may develop in early adult life.

With gradual arterial occlusion the onset is insidious and the symptoms are slowly progressive, but in cases precipitated by thrombosis or embolism the onset may be sudden. In a typical case the patient complains that after walking a certain distance—perhaps a hundred yards or so—he is forced to stop by severe cramp-like pain in the calf, or occasionally in another muscle group. After he has rested for a few minutes the pain disappears and he is able to walk on again for a similar distance. *On examination* there is objective evidence of impaired arterial circulation in the lower limb. The posterior tibial, dorsalis pedis, and popliteal pulses are absent. There may be ischaemic changes in the skin of the foot. Evidence of widespread arterial or cardiac disease is nearly always found on general examination. *Radiographic examination*: Plain radiographs will often show patches of calcification in the walls of several arteries. Arteriographs will demonstrate the site and extent of the arterial occlusion.

Diagnosis. Intermittent claudication is frequently misdiagnosed. Unless a detailed history is obtained, the patient's complaint of

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Diagnosis. Intermittent claudication is frequently misdiagnosed. Unless a detailed history is obtained, the patient's complaint of

"pain in the calf" suggests to the unwary doctor the likelihood of foot strain or "flat foot." Moreover, such a diagnosis may seem to be supported by the finding of flattened arches or deformed feet, common in the elderly. The clues to the correct diagnosis are the typical history and the impairment of the arterial pulses.

Prognosis. The outlook is serious. In progressive cases increasing ischaemia may lead eventually to gangrene of the foot, if the patient does not die first from cardiac disease.

Treatment. Except in rare instances, treatment is rather unsatisfactory. If the nutrition of the limb as a whole is not in danger it is doubtful whether any treatment is worth while. The exceptional cases are those in which there is a localised occlusion, the general state of the arteries being reasonably good. In these circumstances there is a place for excision of the occluded length and restoration of continuity by an artery graft. In cases of severe pain tenotomy of the tendo calcaneus has been tried. It affords relief by reducing the activity of the calf muscles.

If the nutrition of the foot is threatened lumbar ganglionectomy is often worth a trial as a means of improving the circulation in the skin, but it cannot be expected to have a significant effect on the symptoms of claudication.

DISORDERS OF THE ANKLE

PYOGENIC ARTHRITIS OF THE ANKLE

(General description of pyogenic arthritis, p. 31.)

Pyogenic arthritis of the ankle is uncommon. The organisms reach the joint through the blood stream or through a penetrating wound; local spread from a focus of osteomyelitis of the tibia or fibula is rare because the bony metaphyses are entirely extracapsular.

Clinical features. There are pain, swelling, and loss of function of the ankle. There is usually constitutional illness, with pyrexia *On examination* the ankle region is diffusely swollen, the concavities in front of and behind the malleoli being obliterated. The skin over the joint is warmer than normal. Movements of

the ankle are markedly impaired, and painful if forced. *Radiographs* are normal at first. Later, if the infection persists, there is diffuse osteoporosis, often with diminution of the cartilage space. *Investigations :* There is a polymorphonuclear leucocytosis. Aspiration of the joint yields pus, from which the causative organism can usually be identified.

Treatment. This is along the lines suggested for pyogenic arthritis in general. It consists in the systemic and local administration of the appropriate antibiotic drug, with repeated aspiration of the joint and splintage until the infection is overcome.

RHEUMATOID ARTHRITIS OF THE ANKLE

(General description of rheumatoid arthritis, p. 34.)

One or both ankles are often affected by rheumatoid arthritis in common with other joints.

Clinical features. The ankle is painful, diffusely swollen from synovial thickening, and unusually warm. Movements are restricted, and painful if forced. Several other joints are similarly affected. *Radiographs* show no abnormality at first. Later, there is diffuse osteoporosis, and later still there may be diminution of the cartilage space. *Investigations :* The erythrocyte sedimentation rate is raised while the disease is active.

Course. The disease tends to " burn itself out " after months or years of activity, leaving a damaged joint. Secondary degenerative changes may lead to superimposed osteoarthritis.

Treatment. This is along the lines suggested for the disease as a whole (p. 36) There is no specific remedy. Systemic measures comprise drugs such as aspirin, phenyl butazone, or gold salts ; artificial hyperpyrexia ; and cortisone or ACTH (in selected cases). *Local treatment :* In the active phase rest in bed or in a plaster is sometimes required. But in most cases the patient should be encouraged to remain active so far as possible. Physiotherapy is well worth a trial : the most effective methods are short-wave diathermy, active exercises, and hot wax baths. *Operative treatment :* If superimposed osteoarthritic changes lead to severe disablement, arthrodesis of the ankle should be recommended. If the subtalar and midtarsal joints are also severely affected they should be included in the fusion.

TUBERCULOUS ARTHRITIS OF THE ANKLE

(General description of tuberculous arthritis, p. 37.)

Tuberculosis is much less common in the ankle than it is in the hip and knee

Clinical features. The patient is usually a child, but adults are sometimes affected. There are pain and swelling of the ankle, with limp. *On examination* the ankle is diffusely swollen from synovial thickening The overlying skin is warmer than normal. Movements are impaired in both directions—dorsiflexion and plantar-flexion—and cause pain if forced. Movements of the tarsal joints are unaffected A tuberculous lesion is often present elsewhere in the body *Radiographic examination :* The earliest sign, as in other tuberculous joints, is diffuse osteoporosis of the whole area of the joint. If the disease progresses the cartilage space is narrowed or obliterated and the underlying bone is eroded *Investigations :* The erythrocyte sedimentation rate is usually raised. Biopsy of the synovial membrane shows the histological features of tuberculosis.

Treatment. This should be carried out in a country orthopaedic hospital, and should follow the lines advocated for tuberculosis of the hip or knee. A few joints can be saved and restored almost to normal by conservative treatment, but in many cases arthrodesis of the ankle is required after the disease has become quiescent.

OSTEOARTHRITIS OF THE ANKLE

(General description of osteoarthritis, p 41.)

Degenerative destruction of the articular cartilage is less common in the ankle than in the knee, but it may cause serious disability, sometimes necessitating operative treatment

Cause. The essential cause is wear and tear. But there is nearly always a predisposing factor which causes the joint to wear out prematurely. The commonest is irregularity or mal-alignment of the joint surfaces after a fracture. Sometimes articular disease such as " burnt out " rheumatoid arthritis is the primary factor.

Pathology. The articular cartilage is slowly worn away until subchondral bone is exposed This becomes hard and eburnated. At the margins of the joint the bone hypertrophies to form a rim of osteophytes.

Clinical features. The symptoms are pain which slowly increases over months and years, and limp. There is nearly always a history of some previous injury or disease of the ankle. *On examination* the joint is a little thickened from the marginal bony hypertrophy. Movements of the ankle are limited slightly or severely according to the degree of arthritis. *Radiographs* show

Fig. 265
Osteoarthritis of the ankle. Note the marked narrowing of the cartilage space and the subchondral sclerosis.

the usual features of osteoarthritis—narrowing of the cartilage space, a tendency to sclerosis of the bone adjacent to the joint, and hypertrophy (osteophyte formation) at the joint margins (Fig. 265).

Treatment. In mild cases treatment is often unnecessary, for the patient may be willing to accept the disability when the nature of the trouble has been explained. When treatment is called for, conservative measures should be tried first if the disability is only moderate. Physiotherapy by short-wave diathermy, wax baths, and active exercises is usually advised. Such treatment, however, is only palliative, and if the disability increases to the extent of

becoming a serious handicap operation should be undertaken. The only satisfactory operation is arthrodesis.

GOUTY ARTHRITIS OF THE ANKLE

Although gout is commonest in the joints of the great toe it should be remembered that it can occur in the ankle and in other peripheral joints. The general features of gouty arthritis were described in Chapter II (p. 44).

HAEMOPHILIC ARTHRITIS OF THE ANKLE

(General description of haemophilic arthritis, p. 46.)

Haemophilic arthritis is much less common in the ankle than in the knee or elbow, but it should be remembered as an occasional cause of a warm, swollen ankle in boys. There is a spontaneous effusion of blood into the joint. The blood irritates the synovial membrane, which becomes thickened. Joint movements are restricted. The condition may be mistaken for chronic infective arthritis, but there is nearly always a history of previous bleeding incidents, and the clotting time of the blood is usually prolonged. Treatment is by rest in plaster for several weeks until the irritative reaction settles down. Operative treatment should not be contemplated.

CHARCOT'S OSTEOARTHROPATHY OF THE ANKLE

(General description of Charcot's osteoarthropathy, p. 47.)

Although it more often affects the knee, Charcot's osteoarthropathy is well recognised in the ankle. The usual underlying factor responsible for the neurological disturbance is tabes dorsalis Less often, diabetic neuritis is the primary factor.

Clinical features. The patient complains of swelling and instability of the ankle. Pain is usually absent or slight. *On examination* the ankle is swollen, mainly from bony thickening. The range of movement is impaired. The ankle is unstable, allowing abnormal lateral movements. The clinical features of the underlying condition (tabes dorsalis or diabetic neuritis) will be apparent. *Radiographs* show disorganisation of the joint. The articular surfaces are irregular and there may be much new bone about the joint.

Treatment. In many cases protection by a firm surgical boot, reinforced if necessary by double below-knee steels, is all that is required. Occasionally arthrodesis of the ankle may have to be considered. Appropriate treatment must be given for the underlying neurological disorder.

RECURRENT SUBLUXATION OF THE ANKLE

Severe injury to the lateral ligament of the ankle may cause persistent instability with recurrent attacks of "giving way." The causative injury is always a forced inversion.

Pathology. The lateral ligament is torn and fails to heal. With sudden unguarded inversion stresses the ankle joint "opens up" at the lateral side, the talus tilting within the tibio-fibular mortice (Fig. 266)

Clinical features. The patient complains that the ankle "goes over" at frequent intervals, often causing him to fall. Each incident is accompanied by pain at the lateral side of the ankle. There is always a history of previous severe injury, followed by much swelling and extensive bruising at the lateral side of the joint. *On examination* there is often some oedema around the ankle. There is tenderness over the site of the lateral ligament. The normal ankle movements—dorsiflexion and plantar-flexion—are unchanged, but abnormal mobility is present as shown by the fact that the heel can be inverted passively through a greater range than the normal permitted by the subtalar joint. Moreover, when the heel is fully inverted a dimple or depression of the skin is often visible in front of the lateral malleolus, where the soft tissues have been "sucked" into the gap created between tibia and talus. *Radiographic examination :*

FIG. 266
Tilting of the talus in the ankle mortice under adduction stress, an indication of torn lateral ligament.

Routine radiographs show no abnormality. Antero-posterior films must be taken while the heel is held fully inverted. If the lateral ligament is torn or lax the talus will be shown tilted away from the tibio-fibular mortice at the lateral side through 20 or 30 degrees or more (Fig. 266).

Treatment. If the disability is slight it may be sufficient to strengthen the evertor muscles (mainly the peronei) by exercises, to enable them to control the ankle more efficiently. But if disability is severe operation is required. A new lateral ligament is constructed from the peroneus brevis tendon.

DISORDERS OF THE FOOT

CONGENITAL CLUB FOOT

(Talipes equino-varus)

The rather vague term " club foot " has come to be synonymous in the minds of most surgeons with the most important congenital deformity of the foot— talipes equino-varus The less serious form of club foot, talipes calcaneo-valgus, will be considered later under that title

Cause. In most cases a defect of foetal development is responsible. Minor degrees of the deformity may possibly be explained by prolonged mal-position of the foetal foot in the uterus, but this cannot be accepted as the usual cause

Pathology. The soft tissues at the medial side of the foot are under-developed and shorter than normal The foot is adducted and inverted at the subtalar, midtarsal, and anterior tarsal joints, and is held in equinus (plantar-flexion) at the ankle. In the absence of early effective treatment the developing tarsal bones become misshapen, perpetuating the deformity In many cases the calf and peroneal muscles are under-developed.

Clinical features. The deformity is much commoner in boys than in girls. (Contrast congenital dislocation of the hip, which is much commoner in girls.) One or both feet may be affected. When the infant is born it is noticed that the foot is turned inwards so that the sole is directed medially (Fig. 267). The deformity, to be more precise, consists of three elements · 1) inversion (twisting inwards) of the foot, 2) adduction (inward deviation) of the

orefoot relative to the hindfoot; and 3) equinus (plantar-lexion). The foot cannot be pushed passively through the normal range of eversion and dorsiflexion.

Diagnosis. It is not sufficient, for purposes of diagnosis, that the foot rests in the position described, for often the feet of normal infants tend to lie naturally in a somewhat inverted position. The criterion for the diagnosis of club foot is that the deformity cannot readily be corrected and over-corrected to bring the foot into eversion and dorsiflexion. It should be remembered that in normal infants under one year old it is possible to evert and dorsiflex the foot far enough to bring the little toe into contact with the shin.

Fig. 267
Congenital club foot. The typical deformity.

Treatment in fresh cases. Treatment must be begun immediately after birth—certainly not more than one week later. The longer the delay before treatment is begun the smaller is the prospect of complete cure. If efficient treatment is delayed for more than three months restoration to normal is impossible.

The principles of treatment are: 1) to correct and over-correct the deformity by repeated firm manual pressure; and 2) to hold the foot in the over-corrected position until there is no longer a tendency for the deformity to recur (usually from three to twelve months). 3-12

Correction of deformity. The deformity is corrected by firm manual pressure without anaesthesia (Fig. 268). The adduction and inversion are corrected first, and finally the equinus. According to the severity of the deformity, it may be possible to correct it fully at the first manipulation, or as many as six or eight manipulations (at weekly intervals) may be required.

Maintenance of correction. Two methods are available for holding the foot in the corrected position between manipulations: 1) a plaster-of-Paris case; and 2) metal splints as used by Denis

Routine radiographs show no abnormality. Antero-posterior films must be taken while the heel is held fully inverted. If the lateral ligament is torn or lax the talus will be shown tilted away from the tibio-fibular mortice at the lateral side through 20 or 30 degrees or more (Fig. 266).

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Clinical features. The deformity is much commoner in boys than in girls. (Contrast congenital dislocation of the hip, which is much commoner in girls.) One or both feet may be affected. When the infant is born it is noticed that the foot is turned inwards so that the sole is directed medially (Fig. 267). The deformity, to be more precise, consists of three elements: 1) inversion (twisting inwards) of the foot; 2) adduction (inward deviation) of the

clamped rigidly to a cross bar with the feet rotated outwards (Figs. 270 and 271). To remain effective, the splints must be re-applied every day or at least on alternate days.

FIG. 270

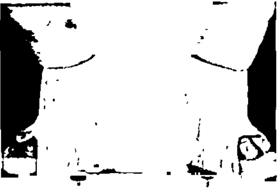

FIG 271

Denis Browne splints for congenital club foot. The feet are held to the sole plates by adhesive strapping, as shown in Figure 271.

Treatment in neglected or relapsed cases. Repeated manipulation and retention in plaster can produce worth-while improvement in children of up to 3 years of age. If significant *deformity is still present after the age of 3, operative treatment is required.* It should be appreciated that in these late cases no method of treatment—whether conservative or operative—is capable of restoring the foot to normal. The most that can be done is to restore a plantigrade foot.

Types of operation · In children of from 3 to 12 years three operations are to be considered: 1) division of the short soft tissues at the medial side of the foot, thereafter forcing the foot into a plantigrade position and immobilising it in plaster for three months; 2) transfer of the tendon of tibialis anterior or tibialis posterior to the lateral side of the foot to supplement the action of the evertor muscles; and 3) lengthening of a short tendo calcaneus.

In children over the age of 12 resort must be had to operation upon the bones a wedge of bone of appropriate size (with base dorso-laterally) is removed from the tarsus so that when the resulting gap is closed the foot is plantigrade. This operation is not recommended for children under 12 because it may impair the growth of the foot.

Browne. Retention in a plaster is preferred, because it holds the foot in the over-corrected position more efficiently and for a longer period than metal splints. The plaster must extend to the upper

FIG 268
Correcting a club-foot deformity by manual pressure without anaesthesia.

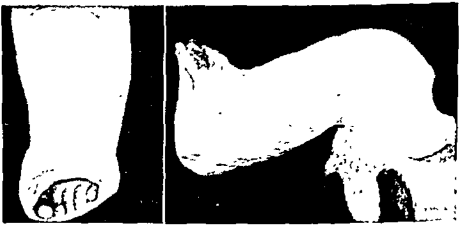

FIG 269
Plaster for maintaining correction in congenital club foot. It is essential to include the thigh, with the knee flexed to a right angle.

thigh, with the knee flexed 90 degrees (Fig. 269); otherwise the infant is able to draw the foot up inside the plaster. The plaster must be changed every week at first, but the interval can be extended to two and then three weeks as the child grows larger. In the Denis Browne method both feet are fixed to the metal splints by strips of adhesive strapping, and the splints are then

ACCESSORY BONES IN THE FOOT

Many accessory bones have been described in the foot, but most are of little or no practical importance. The commonest is the **os trigonum**, which lies immediately behind the talus, upon the upper surface of the tuberosity of the calcaneum (Fig. 273). It does not cause symptoms. It may be confused

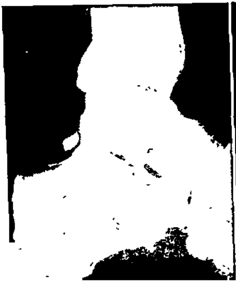

FIG. 273
Os trigonum.

FIG 274
Os tibiale externum.

with a fracture of the talus. The only tarsal accessory bone that is frequently responsible for symptoms is the **os tibiale externum** (accessory navicular bone) (Fig. 274). This lies medial to the navicular, and forms a well marked prominence at the inner border of the foot which may become painful and tender from the pressure of the shoe. If the symptoms justify operation the accessory bone should be removed.

PES CAVUS

In pes cavus or "hollow foot" the longitudinal arch of the foot is accentuated.

Cause. In most cases the deformity has a congenital basis. It sometimes runs in families. In other cases there is an underlying neurological disorder causing muscle imbalance. For instance,

CONGENITAL TALIPES CALCANEO-VALGUS

This is the opposite deformity to talipes equino-varus The foot is everted and dorsiflexed. It is a much less serious deformity than talipes equino-varus because it responds more readily to treatment.

Cause. The cause is unknown. In some cases it may simply be a postural deformity, from folding of the foot against the shin for a long time in intra-uterine life.

Clinical features. One or both feet may be affected. The foot rests in a position of eversion and dorsiflexion, so that its dorsum lies almost in contact with the shin (Fig. 272). Tightness of the dorso-lateral soft tissues prevents the foot's being brought down into inversion and equinus, though with steady pressure a fair degree of correction can usually be obtained.

Treatment. The problem of treatment is much simpler than it is in talipes equino-varus. In most cases the deformity will respond to repeated manual stretching by the parents, who should be carefully instructed how to coax the foot into the over-corrected position of inversion and equinus by steady pressure upon the dorsum of the foot. The manipulations should be begun immediately after birth and should be carried out several times a day.

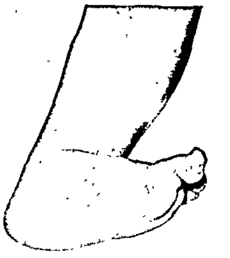

Fig 272
Congenital talipes calcaneo-valgus
The typical deformity

If calcaneo-valgus deformity still persists when the child is a month old more intensive supervision is required. The surgeon should gently over-correct the deformity as far as possible by manipulation without anaesthesia, thereafter applying a plaster with the foot in the over-corrected position. The plaster is changed weekly until a full range of inversion and equinus is regained. At that stage the plaster can be discarded without fear of relapse.

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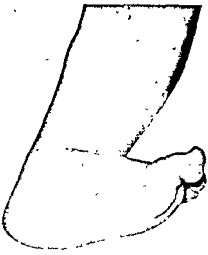

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heads; 2) tenderness over the deformed toes from pressure against the shoe; and 3) pain in the tarsal region from osteoarthritis of the tarsal joints. *On examination* the deformity is characteristic and easily recognised (Fig. 275). The longitudinal arch is high; the forefoot is thick and splayed; the toes are clawed; the metatarsal heads are prominent in the sole. Callosities beneath the metatarsal heads indicate that they take excessive weight. The toes cannot be straightened at will by the patient, nor can they be pressed firmly upon the ground to take a share in weight-bearing. There may also be tender callosities or abrasions where the tops of the toes have rubbed against the shoes.

Treatment. Many patients require no treatment. Those with mild symptoms can often be relieved by regular chiropody and by the provision of a soft sponge-rubber pad beneath the metatarsal heads to distribute the weight more widely.

Patients with severe symptoms require operative treatment. The nature of the operation should depend upon the cause of the main symptoms.

Operations on the toes. When local pressure upon the toes or beneath the metatarsal heads is the main factor the shape of the foot should be improved so far as possible without interfering with the tarsal joints. Considerable improvement can often be effected simply by straightening the curled toes by arthrodesis of all the interphalangeal joints or by transplanting the long flexor tendon into the extensor expansion.

Operations on the soft tissues of the sole. In severe cases arthrodesis of the toes should be supplemented by the Steindler muscle-slide operation, in which the taut ligamentous tissues in the sole of the foot are detached from the calcaneum and allowed to slide forwards as the height of the arch is reduced by strong manual force. After operation the correction is held by immobilising the foot in plaster for two months.

Operations on the tarsal joints When osteoarthritis of the tarsal joints is the main cause of the symptoms arthrodesis of the affected joints (usually the subtalar, calcaneo-cuboid, talo-navicular, and naviculo-cuneiform) is required. At the same time the deformity is corrected by excising a wedge of bone, base upwards, from the metatarsal region. When necessary, this operation may be combined with operations to straighten the toes.

it is sometimes associated with spina bifida, or it may follow poliomyelitis.

Pathology. The soft tissues at the sole of the foot are unduly short, so that they seem to act like a bowstring approximating the anterior and posterior pillars of the longitudinal arch. The tarsal bones themselves assume an abnormal wedged shape to conform with the deformity. Secondary changes affect the toes, which become clawed, with hyperextension at the metatarso-phalangeal

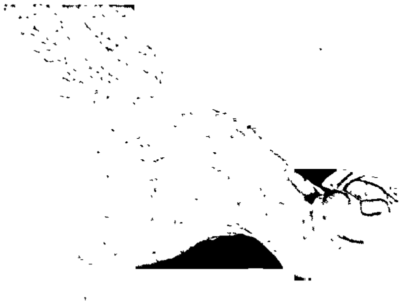

FIG. 275
Pes cavus. Typical deformity with high arch, clawed toes, and prominence of the metatarsal heads in the sole.

joints and fixed flexion deformity at the proximal and distal interphalangeal joints. This clawing makes the toes almost functionless, so that they are unable to share in weight-bearing. Consequently excessive weight falls upon the metatarsal heads on walking or standing, and hard callosities form in the underlying skin. The mal-alignment of the tarsal joints predisposes to the later development of osteoarthritis.

Clinical features. The deformity often becomes evident in childhood. It may affect one foot or both. In some cases the symptoms are negligible. *When symptoms do arise they may take three forms*: 1) painful callosities beneath the metatarsal

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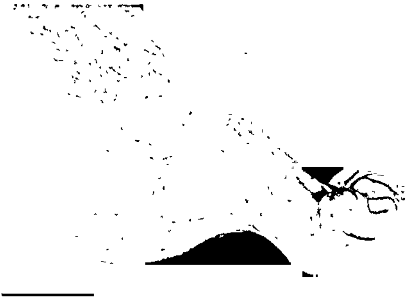

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arthritis. In children with markedly valgus feet a common complaint of the parents is that the uppers of the shoes persistently bulge over on the inner side and wear out quickly.

Treatment. In children under 3 years old treatment is not required. In children over 3 the accepted method of treatment is to tilt the shoe slightly to the lateral side by inserting a wedge, base medially, between the layers of the heel (not the sole) (Fig. 277). This helps to overcome the valgus twist and reduces the bulging-over of the uppers at the medial side. In older children this treatment is supplemented by a course of supervised exercises to strengthen the intrinsic muscles of the foot.

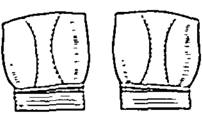


FIG. 277

Shoes drawn from behind, to illustrate medial heel wedges

In adults treatment is not needed unless symptoms are present. If symptoms are due to superimposed foot strain, as is usually the case, treatment appropriate to that condition is advised. At first reliance is placed upon a course of exercises and electrical (faradic) stimulation, under the supervision of a physiotherapist, to strengthen the intrinsic muscles of the foot and the calf muscles. If these measures fail to give relief the advisability of fitting an arch support should be considered. Supports are seldom of benefit when the foot is completely flat, but they often afford relief when the longitudinal arch is diminished but not lost. They should always be made from a plaster model of the foot. In persistently troublesome cases manipulation of the feet under an anaesthetic is worth a trial.

If the symptoms in a case of long-established flat foot are ascribed to superimposed osteoarthritis of the tarsal joints, treatment should be directed against the arthritis (p. 388).

FOOT STRAIN

Foot strain is a subacute or chronic strain of the tarsal ligaments.

Cause. In feet previously normal foot strain is caused by excessive standing by a person unaccustomed to it. In feet whose intrinsic structure is already impaired (for example, by flattened arches)

PES PLANUS

(Flat foot; valgus foot)

In this common condition the longitudinal arch of the foot is reduced so that, on standing, its medial border is close to, or in contact with, the ground (Fig. 276). It is usually associated with some degree of twisting outwards of the foot on its longitudinal axis (*eversion or valgus deformity*).

Fig 276
Pes planus. Marked flattening of the longitudinal arch, with valgus deformity seen well from behind (inset)

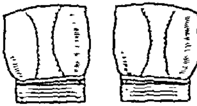
Cause. This is unknown. In most cases it probably has a congenital basis.

Pathology. All infants have flat feet for a year or two after they begin to stand. When the deformity persists into adult life it becomes a permanent structural defect, the tarsal bones being so shaped that when articulated they tend to form a straight line rather than an arch.

Clinical features. Flat feet are always symptomless in children and often also in adults. But they are more liable than are normal feet to suffer foot strain (p. 387) and, in later life, tarsal osteo-

arthritis. In children with markedly valgus feet a common complaint of the parents is that the uppers of the shoes persistently bulge over on the inner side and wear out quickly.

Treatment. In children under 3 years old treatment is not required. In children over 3 the accepted method of treatment is to tilt the shoe slightly to the lateral side by inserting a wedge, base medially, between the layers of the heel (not the sole) (Fig. 277). This helps to overcome the valgus twist and reduces the bulging-over of the uppers at the medial side. In older children this treatment is supplemented by a course of supervised exercises to strengthen the intrinsic muscles of the foot.



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Cause. In feet previously normal foot strain is caused by excessive standing by a person unaccustomed to it. In feet whose intrinsic structure is already impaired (for example, by flattened arches)

strain often arises from the ordinary amount of standing demanded in everyday life.

Pathology. In the normal foot the tarsal ligaments are protected from strain by the action of muscles. Thus a force tending to deform the longitudinal arch is resisted by the muscles responsible for maintaining the arch, and not by the tarsal ligaments. When the muscles are unequal to their task the stress falls upon the ligaments, which become strained. The mechanical irritation of recurrent strains leads to chronic inflammatory changes in and around the ligaments. This inflammation is responsible for the characteristic aching after activity.

Clinical features. During and after prolonged walking or standing there is aching pain either in the foot alone or in the foot and calf. Pain in the foot is felt mainly in the midtarsal region : commonly it extends along the under side of the medial border of the foot and along the dorsum of the foot. Calf pain is usually posterior, but it may be felt in the anterior leg muscles. *On examination* the foot is often of normal shape and appearance. In other cases there is flat foot or other deformity. In severe cases there is tenderness over the tarsal ligaments in the sole of the foot.

Diagnosis. Foot strain is easily confused with intermittent claudication, the symptoms of which are in some respects similar. With careful enquiry and clinical examination there should be no difficulty in making the distinction. The history of intermittent claudication is highly characteristic, and the absence of palpable pulses at the foot (and usually also at the knee) is important confirmatory evidence.

Treatment. In mild cases it is sufficient to curtail the amount of standing and walking, and to arrange a course of supervised exercises and electrical (faradic) stimulation to strengthen the muscles of the leg and foot

In severe cases more complete rest is required. This is provided either by recumbency or by immobilisation in a plaster, retained for four weeks.

OSTEOARTHRITIS OF THE TARSAL JOINTS

Osteoarthritis may affect any of the tarsal joints, but in practice it is seen most often in the subtalar and midtarsal joints. It seldom arises primarily : there is nearly always a predisposing

factor such as previous fracture or disease involving the joint surfaces (especially fractures of the calcaneum), or mal-alignment of the tarsal bones (as in severe flat foot or severe pes cavus)

Pathology. This is the same as that of osteoarthritis elsewhere. The articular cartilage is worn away and the joint margins are hypertrophied, forming a fringe of osteophytes.

Clinical features. The main symptom is pain, which gradually increases over months or years and finally leads to limp and impaired capacity for walking. Pain is localised fairly accurately to the particular joint or joints affected. A history will usually be obtained of previous injury, disease, or deformity of the foot. *On examination* movements of the affected joint are impaired. *Radiographs* confirm the diagnosis and may give an indication of the primary underlying cause.

Treatment. In mild cases treatment is not required, especially if the patient can curtail his activities. When the disability is troublesome more active measures are needed. Conservative treatment is only palliative and cannot abolish the pain completely, but it is usually worth a trial. It should take the form of short-wave diathermy or hot wax baths, muscle exercises, and support from a crepe bandage.

If conservative treatment fails to give adequate relief operation must be considered. The only effective method is by arthrodesis of the affected joint or joints.

OTHER FORMS OF ARTHRITIS OF THE TARSAL JOINTS

Like all true synovial joints, the tarsal joints are liable to any of the recognised forms of arthritis, including pyogenic arthritis, rheumatoid arthritis, tuberculous arthritis, gouty arthritis, and Charcot's osteoarthropathy. Of these, the only one that is at all common is rheumatoid arthritis.

OSTEOCHONDRITIS OF THE NAVICULAR BONE

(Köhler's disease)

The general subject of osteochondritis was discussed in Chapter II (p 77). The growing navicular bone is one of its best recognised sites. The developing nucleus of the bone is temporarily

softened and usually becomes compressed by the mechanical forces entailed in walking. A disturbance of blood supply is possibly a causative factor.

Pathology. The pathology is like that of osteochondritis of other growing bony nuclei (Fig. 60, p. 79). The bone loses its normal trabecular structure and becomes granular, and sometimes

FIG 278

FIG. 279

Köhler's osteochondritis of the navicular bone. Figure 278—Early stage Bony nucleus dense and flattened antero-posteriorly Figure 279—Two years later Bone texture restored ; slight residual flattening

fragmented. After about two years the normal bone structure is restored Some deformation of the bone remains, but the growing foot seems to adapt itself to the altered shape and little or no disability persists Nevertheless there is probably a predisposition to the later development of osteoarthritis of the talo-navicular and naviculo-cuneiform joints

Clinical features. Kohler's disease is confined to children of about 3 to 5 years. The child complains of pain in the midtarsal part of the foot and is noticed to limp *On examination* there is no obvious change in the shape of the foot. There is tenderness on firm palpation over the navicular region. There may be some restriction of midtarsal movements with pain on forcing, but these signs are slight and sometimes absent *Radiographs* are diagnostic The bony nucleus of the navicular bone appears squashed from before backwards ; it is denser than normal, and often has a

fragmented appearance (Fig. 278). [illegible] two-year span of the disease show the gradual [illegible] of the bone changes. After a stage of [illegible] a few months after the onset there is gradual [illegible] the normal bone texture is restored (Fig. 279).

Treatment. Theoretically it might be wise to [illegible] weight-bearing for the whole of the two-year period of the [illegible] as in Perthes' osteochondritis at the hip. In practice, however, this is not necessary, for good results follow less drastic [illegible]. It is usually sufficient simply to immobilise the foot in a walking plaster for two months.

OSTEOCHONDRITIS OF THE CALCANEAL APOPHYSIS

(Sever's disease; calcaneal apophysitis)

This is another example of osteochondritis occurring in the foot. It will be described in the section on painful heel (p. [illegible]).

OSTEOCHONDRITIS OF A METATARSAL HEAD

(Freiberg's disease)

This is the third example of osteochondritis in the foot. It will be described in the section on disorders of the toes (p. 408).

PAINFUL HEEL

The causes of painful heel are conveniently classified according to the site of the pain (Fig. 280).

PAIN WITHIN THE HEEL
- Disease of the calcaneum
  - (osteomyelitis; tumour; Paget's disease)
- Arthritis of the subtalar joint

PAIN BEHIND THE HEEL
- Rupture of the tendo calcaneus (p. 366)
- Calcaneal paratendinitis
- Post-calcaneal bursitis
- Osteochondritis of the calcaneal apophysis

PAIN BENEATH THE HEEL
- Tender heel pad
- Plantar fasciitis

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Pathology. The pathology is like that of osteochondritis of other growing bony nuclei (Fig. 60, p. 79). The bone loses its normal trabecular structure and becomes granular, and sometimes

Fig. 278

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Kohler's osteochondritis of the navicular bone. Figure 278—Early stage Bony nucleus dense and flattened antero-posteriorly. Figure 279—Two years later Bone texture restored ; slight residual flattening

fragmented. After about two years the normal bone structure is restored. Some deformation of the bone remains, but the growing foot seems to adapt itself to the altered shape and little or no disability persists. Nevertheless there is probably a predisposition to the later development of osteoarthritis of the talo-navicular and naviculo-cuneiform joints

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be termed paratendinitis rather than tenosynovitis, for there is no true synovial sheath

Clinical features. The patient is usually an active young adult. There is pain in the region of the tendo calcaneus, made worse by activities such as running or dancing. *On examination* there is tenderness on palpation between finger and thumb deep to the tendon (Fig. 280) and there is slight local thickening in this region The tendon itself is of normal size and consistency

Treatment. In most cases relief is afforded by resting the ankle in a below-knee walking plaster for four weeks If not, the loose connective tissue surrounding and deep to the tendon should be excised.

POST-CALCANEAL BURSITIS

This is the commonest cause of pain behind the heel. It is often a cause of troublesome disability in young women.

Pathology. An adventitious bursa forms at the back of the heel, between the tuberosity of the calcaneum and the skin Repeated friction against the back of the shoe leads to chronic inflammation and thickening of the walls of the bursa, and the sac may be distended with fluid.

Clinical features. There is troublesome tenderness where the swelling is in contact with the shoe (Fig. 280). The symptoms are aggravated by walking, and they tend to be worse in winter than in summer. *On examination* there is an obvious gristly prominence at the back of the heel ; the overlying skin is thickened and may be red

Treatment. In mild or recent cases the symptoms can be controlled by protecting the back of the heel with a double layer of elastic adhesive strapping and by wearing shoes with soft backs. If these measures fail the bursa should be excised and any sharp prominence of the underlying bone removed

OSTEOCHONDRITIS OF THE APOPHYSIS OF THE CALCANEUM

(Sever's disease; calcaneal apophysitis)

This harmless condition is probably analogous to Osgood-Schlatter's disease of the tibial tubercle. It is characterised by pain in the region of the calcaneal apophysis, which may be slightly enlarged It occurs only in children, during the period of active growth of the apophysis.

DISEASE OF THE CALCANEUM

The calcaneum is subject to all types of infection of bone, the commonest being pyogenic infection (osteomyelitis) and tuberculosis. Rarely, it is the seat of a benign or malignant bone tumour.

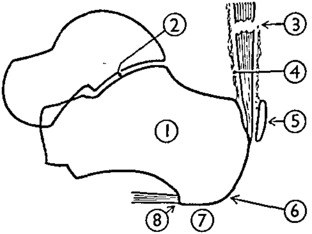

FIG. 280

Eight causes of painful heel, with site of pain. 1 Disease of the calcaneum. 2. Arthritis of the subtalar joint 3. Ruptured calcaneal tendon. 4 Calcaneal paratendinitis 5 Post-calcaneal bursitis. 6. Osteochondritis of the calcaneal apophysis 7. Tender heel pad 8. Plantar fasciitis

It may also be affected by other disorders of bone, such as Paget's disease All of these conditions were described in Chapter II, and no special description is required here.

ARTHRITIS OF THE SUBTALAR JOINT

The commonest type of arthritis in the subtalar joint is osteoarthritis secondary to a fracture of the calcaneum (p. 388). The joint is occasionally subject to other forms of arthritis, such as pyogenic arthritis, rheumatoid arthritis, tuberculous arthritis, and gout.

CALCANEAL PARATENDINITIS

(Calcaneal "tenosynovitis")

The calcaneal tendon is surrounded by loose connective tissue, or paratenon, which allows gliding movements. Rarely, this becomes inflamed from excessive friction The condition should

PLANTAR FASCIITIS

In this condition, which is believed to be inflammatory, there is pain beneath the anterior part of the calcaneum.

Pathology. The lesion affects the fascia and soft connective tissue at the site of attachment of the plantar fascia to the inferior aspect of the tuberosity of the calcaneum. The precise cause of the inflammation is uncertain; it may be a toxic reaction set up by the presence of an infective process elsewhere in the body.

Clinical features. The complaint is of pain beneath the heel on standing or walking; the pain may extend forwards into the sole. The disability is sometimes severe. There is often a history of a recent infective lesion elsewhere (for example, sore throat or urethritis). *On examination* there is marked tenderness over the site of attachment of the plantar fascia to the calcaneum. The site of tenderness is farther forward than it is in tender heel pad (Fig. 280) *Radiographs* usually show no abnormality. A sharp spur projecting forwards from the tuberosity of the calcaneum is sometimes found; but its significance is doubtful, for such spurs may be present in patients without heel symptoms. Nevertheless a large spur should be regarded as pathological, especially if the bone appears to be of recent formation.

Treatment. If there is a focus of infection elsewhere in the body appropriate measures must be taken to eliminate it. For the foot itself, conservative treatment usually suffices if continued for long enough, though recovery may be slow. The heel should be protected by a sponge-rubber cushion on an insole, and a course of short-wave diathermy to the tender area should be arranged. Only in exceptionally severe and resistant cases should resort be had to operation. Spurred bone should be removed and the plantar fascia stripped from its posterior attachment.

PAIN IN THE FOREFOOT

(Metatarsalgia)

Pain in the forefoot is one of the commonest orthopaedic complaints There are three main causes, of which the first is the most frequent: 1) anterior flat foot (dropped transverse arch); 2) stress fracture of a metatarsal (" march " fracture); 3) plantar digital neuritis (Morton's metatarsalgia).

The cause is unknown; and though the condition has been labelled osteochondritis there are many who believe that it represents no more than a strain of the attachments of the apophysis.

FIG. 281
Sever's osteochondritis of the calcaneal apophysis. The bony nucleus of the growing apophysis is enlarged, dense, and fragmented

Clinical features. The child is usually between 8 and 13 years old. He complains of pain behind the heel, and a slight limp may be noticed. *On examination* there are swelling and tenderness over the lower posterior part of the tuberosity of the calcaneum (Fig. 280). *Radiographs* often fail to show any alteration from the normal, but there may be slight enlargement of the apophysis, with an appearance of fragmentation (Fig 281).

Treatment. In most cases treatment is not required, for the symptoms will gradually subside spontaneously. If the pain is severe a few weeks' rest in a below-knee walking plaster will afford adequate relief.

TENDER HEEL PAD

This is a distinct clinical condition characterised by pain beneath the hind part of the heel on standing or walking.

Pathology. The site of the tenderness is the tough fibro-fatty tissue beneath the prominent weight-bearing part of the calcaneum. In some cases the lesion is probably no more than a simple contusion; but in most cases injury seems to play no part and it must be assumed that there is a mild inflammation, of uncertain origin.

Clinical features. Pain beneath the heel on standing or walking is the only symptom. *On examination* there is well marked local tenderness on firm palpation over the heel pad (Fig. 280).

Treatment. There is a tendency to slow spontaneous improvement. Recovery may be hastened by providing a sponge-rubber heel cushion on an insole, and by a course of short-wave diathermy to the tender area.

Treatment. In patients under 50 worth-while improvement can usually be gained by a prolonged course of physiotherapy, designed to strengthen the intrinsic muscles by special exercises aided by faradic stimulation In older patients this treatment is seldom effective, and resort must usually be had to a sponge-rubber support to distribute the weight-bearing pressure over a wide area of the metatarsus.

STRESS FRACTURE OF A METATARSAL

("March" fracture, fatigue fracture)

Stress fractures are unusual in that there is no history of violence. The possibility of fracture may therefore be overlooked. A stress fracture of a metatarsal is not a common cause of pain in the forefoot but the possibility of its occurrence should be remembered

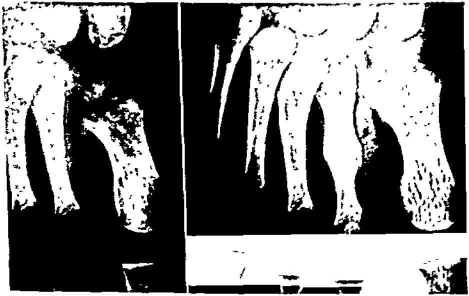

Fig 284 Fig. 285

Stress fracture of second metatarsal Figure 284 shows the initial radiograph, taken a week after the onset of pain. The fracture is seen as a fine crack running across the bone Figure 285 shows the condition two weeks later, in the stage of healing Abundant callus has formed round the site of fracture.

Cause. The fracture is ascribed to long-continued or oft-repeated stress ; it has been likened to the fatigue fractures that sometimes occur in metals

ANTERIOR FLAT FOOT

(Dropped transverse arch)

Permanent flattening of the transverse arch of the foot, with excessive weight-bearing pressure beneath the metatarsal heads, is the commonest cause of metatarsalgia. In most cases the primary cause is *inefficiency of the intrinsic muscles of the foot.*

Pathology. Even in normal feet the transverse arch is only a potential arch, not a constant one. But in the normal state the

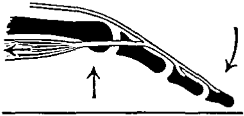

FIG 282

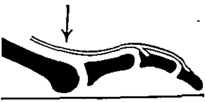

FIG. 283

Figure 282—Diagram to show the normal action of the toes in raising the metatarsal heads from the ground, whereby they share the weight with the metatarsal heads. This action is controlled mainly by the intrinsic muscles. Figure 283—If the intrinsic muscles are inefficient all the weight must be borne by the metatarsal heads

metatarsal heads can be raised from the ground at will by the action of the toes, held straight at the interphalangeal joints by the intrinsic muscles and flexed strongly at the metatarso-phalangeal joints by the intrinsic muscles in conjunction with the long and short flexors (Fig 282). Thus the weight is shared between the metatarsal heads and the toes. If the intrinsic muscles are inefficient the toes are unable to fulfil their important weight-sharing function and all the pressure falls upon the metatarsal heads (Fig. 283). The excessive pressure leads to the formation of callosities beneath the points of pressure.

Clinical features. The main complaint is of pain beneath the forefoot. There may be secondary symptoms from pressure of the shoes upon deformed toes *On examination* the forefoot is often splayed, appearing broader than normal. There are callosities beneath some or all of the metatarsal heads. The toes are often curled or otherwise deformed. The patient is unable to raise the metatarsal heads from the ground by pressing downwards with the toes—an indication that the intrinsic muscles are inefficient.

Treatment. In patients under 50 worth-while improvement can usually be gained by a prolonged course of physiotherapy, designed to strengthen the intrinsic muscles by special exercises aided by faradic stimulation. In older patients this treatment is seldom effective, and resort must usually be had to a sponge-rubber support to distribute the weight-bearing pressure over a wide area of the metatarsus.

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Fig. 284

Fig. 285

Stress fracture of second metatarsal. Figure 284 shows the initial radiograph, taken a week after the onset of pain. The fracture is seen as a fine crack running across the bone. Figure 285 shows the condition two weeks later, in the stage of healing. Abundant callus has formed round the site of fracture.

Cause. The fracture is ascribed to long-continued or oft-repeated stress; it has been likened to the fatigue fractures that sometimes occur in metals.

Pathology. The fracture usually affects the shaft of the second or third metatarsal. It is no more than a hair-line crack, and there is no displacement of the fragments. In the process of healing a large mass of callus may form around the bone at the site of fracture.

Clinical features. The complaint is of severe pain in the forefoot on walking. The onset is rapid but the patient cannot ascribe it to an obvious cause. Enquiry usually reveals, however, that he has done an unusual amount of walking a day or two before the onset. *On examination* there is swelling at the dorsum of the forefoot, with marked and well localised tenderness over the affected metatarsal. *Radiographs* at first show only a faint hair-line crack which is easily overlooked (Fig. 284). But after a week or two the callus surrounding the fracture is clearly visible (Fig. 285).

Treatment. The fracture heals spontaneously, so treatment is purely symptomatic. In some cases no treatment is needed; but if pain is severe immobilisation in a below-knee walking plaster for four weeks is advised.

PLANTAR DIGITAL NEURITIS

(Morton's metatarsalgia; interdigital neuroma)

This condition, which is primarily an affection of a digital nerve, is characterised by metatarsal pain combined with a radiating pain in the third and fourth toes.

Pathology. The underlying lesion is a fibrous thickening or "neuroma" of the digital nerve of the 3-4 cleft just proximal to its point of division into terminal branches It takes the form of a fusiform swelling about three-quarters of an inch in length, surrounding the nerve as it lies in the space between the heads of the third and fourth metatarsals (Fig. 286) Occasionally the nerve to the 2-3 cleft is the one affected. The cause of the fibrous thickening is uncertain

Clinical features. The patient is often a woman of middle age. She complains of pain in the forefoot on standing or walking A characteristic feature is that the pain, arising in the metatarsal region, radiates forwards into the contiguous sides of the third and fourth toes, or to the fourth toe alone. Rarely, the cleft between the second and third toes is affected. Patients often state

that they can get rid of the pain by taking the shoe off and squeezing or manipulating the forefoot. *On examination* the forefoot is often splayed, as in anterior flat foot. Sometimes a painful click can be elicited by compressing the metatarsal heads from side to side, and upward pressure on the sole between the third and fourth metatarsal heads is painful.

Diagnosis. This depends mainly upon the typical history.

Treatment. The patient should try first the effect of wearing a sponge-rubber metatarsal pad to support the anterior arch. If this fails to relieve the symptoms operative excision of the thickened segment of the nerve is recommended.

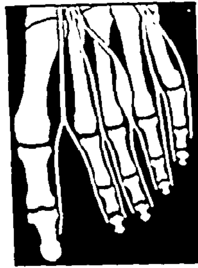

FIG. 286

Fibrous thickening of an interdigital nerve, as found in plantar digital neuritis (Morton's metatarsalgia). The "neuroma" is usually in the 3-4 cleft

PLANTAR WART

(Verruca plantaris)

Plantar warts may occur in any part of the sole of the foot, including the under surface of the heel. They are like warts elsewhere, except that they do not project beyond the skin surface. They are entirely distinct from plantar callosities, which are simply localised thickenings of the skin at points of excessive pressure.

Cause. The exact cause is unknown. A virus infection has been thought responsible.

Pathology. A wart is a simple papilloma, growing outwards from the basal layers of the skin. It is prevented by the pressure of weight-bearing from projecting much beyond the skin surface. But the surrounding skin is thickened, so the wart may have a total depth of up to a quarter of an inch (Fig. 288). It slowly enlarges, but seldom reaches a diameter of more than three-eighths of an inch.

Clinical features. The chief complaint is of severe localised pain on walking. *On examination* the skin surrounding the wart is

Pathology. The fracture usually affects the shaft of the second or third metatarsal. It is no more than a hair-line crack, and there is no displacement of the fragments. In the process of healing a large mass of callus may form around the bone at the site of fracture.

Clinical features. The complaint is of severe pain in the forefoot on walking. The onset is rapid but the patient cannot ascribe it to an obvious cause. Enquiry usually reveals, however, that he has done an unusual amount of walking a day or two before the onset. *On examination* there is swelling at the dorsum of the forefoot, with marked and well localised tenderness over the affected metatarsal. *Radiographs* at first show only a faint hair-line crack which is easily overlooked (Fig. 284). But after a week or two the callus surrounding the fracture is clearly visible (Fig. 285).

Treatment. The fracture heals spontaneously, so treatment is purely symptomatic. In some cases no treatment is needed; but if pain is severe immobilisation in a below-knee walking plaster for four weeks is advised

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toes from taking their proper share in weight-bearing (anterior flat foot, p. 396). They are also common beneath the base of the fifth metatarsal in patients who for any reason walk with the foot inverted.

Callosities on the toes often take the form of localised thickenings, when they are termed corns. They are caused by pressure against the shoe, and they are especially common when the dorsum of a toe is made unduly prominent by a fixed flexion deformity, as in hammer toe.

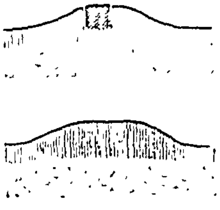

FIG. 288
Diagrammatic section of plantar wart (*top*) and callosity (*bottom*).

Treatment. The treatment is mainly that of the underlying condition. Palliative measures include paring of the excess epidermis (preferably by a chiropodist), and sponge-rubber padding to distribute the weight-bearing pressure over a wider area.

GANGLION

Ganglia are common on the dorsum of the foot and around the ankle. They are similar in all respects to the ganglia that occur at the back of the wrist and hand. They consist of thin-walled sacs filled with glairy viscous fluid, and the fibrous wall is usually connected deeply with a ligament, tendon sheath, or joint capsule. Clinically, a ganglion appears as a fluctuant subcutaneous swelling, which may be either soft or tense. If it causes trouble it should be excised.

DISORDERS OF THE TOES

HALLUX VALGUS

In hallux valgus the great toe is deviated laterally at the metatarso-phalangeal joint. It is common in women past middle age.

Cause. In a few cases congenital factors are responsible. But in

thickened and therefore raised. The wart, a little darker in colour and with a mosaic surface, is seen in the centre of the raised area Its edge is clearly demarcated from the surrounding skin : this can be discerned easily if the skin is stretched away from the wart, when a tiny cleft becomes apparent between wart and skin (Fig. 287). There is always marked local tenderness on pressure over the wart.

FIG. 287
Plantar wart. Note the clearly circumscribed outline and the slight cleft when the skin is stretched away from the wart

Diagnosis. The main difficulty is to distinguish warts from callosities. Warts occur anywhere on the sole, callosities only over points of pressure. Warts are also much more tender than callosities. But the most reliable distinguishing feature is that a wart has a mosaic surface and a clearly defined margin with a potential cleft between it and the skin, whereas a callosity blends imperceptibly with the surrounding normal skin (Fig. 288).

Treatment. The wart should be excised and the base lightly cauterised.

CALLOSITIES

A callosity is simply a localised thickening of the skin in response to abnormal pressure It is nearly always secondary to a pre-existing disorder of the foot.

Plantar callosities are callosities on the sole of the foot They occur under a prominent bone They are commonest beneath the metatarsal heads when deficient intrinsic muscles prevent the

favourite operation is the Keller arthroplasty, the object of which is to create a flail, freely movable false joint between the first metatarsal and the proximal phalanx, with correction of the malalignment. This is done by excising the proximal two-thirds of

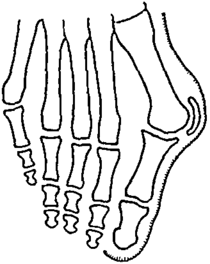

FIG. 290

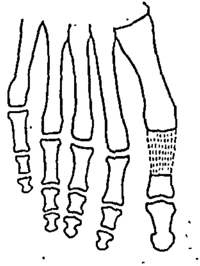

FIG. 291

Hallux valgus Figure 290—Diagram showing the prominent metatarsal head with overlying bunion, and osteoarthritis from mal-alignment of the joint. Figure 291—Excision arthroplasty (Keller's operation): removal of the proximal two-thirds of the proximal phalanx The resulting gap fills with flexible fibrous tissue

the proximal phalanx so that a gap of about half an inch is left between the two bones (Fig. 291). The space fills with rubbery fibrous tissue, which, by virtue of its flexibility, allows a reasonable range of joint movement. The Mayo operation is the same in principle: the head of the first metatarsal is excised instead of the base of the phalanx. A third method, which some surgeons prefer, is to arthrodese the joint in a position of slight extension.

HAMMER TOE

The term hammer toe denotes a fixed flexion deformity of an interphalangeal joint.

Cause. Presumably an imbalance of the delicate arrangement of flexor and extensor tendons is responsible; but the precise explanation of its occurrence is unknown

most the deformity is caused by the toe being persistently forced laterally by enclosure in tight stockings and narrow pointed shoes. The wearing of high heels favours the development of hallux valgus by forcing the forefoot into the narrow pointed part of the shoe.

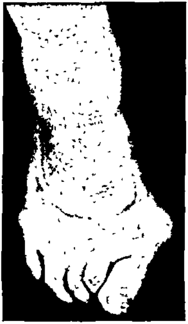

FIG 289

Hallux valgus A typical example There is also fixed flexion deformity of the adjacent toe

Pathology. At first the angular deformity at the joint is the only abnormality. Later, two secondary changes occur. One is the formation of a thick-walled bursa (bunion) over the medial prominence of the joint; this may become inflamed, occasionally with suppuration. The second, a later development, is osteoarthritis of the metatarso-phalangeal joint consequent upon its mal-alignment (Fig. 290).

Clinical features. The patient is nearly always a woman who is usually at or past middle age when she seeks advice. The early symptoms arise from tenderness over the bunion from pressure against the shoe. There is also difficulty in getting comfortable footwear Later, additional symptoms arise from osteoarthritis of the metatarso-phalangeal joint *On examination* the deformity is obvious at a glance (Fig 289). The skin over the prominent joint is hard, reddened, and tender. Often a thick-walled bursa can be felt, and occasionally it is distended with fluid. In relatively early cases metatarso-phalangeal joint movements are free and painless, but in severe cases of many years' duration the secondary osteoarthritis makes movement limited and painful

Treatment. In mild cases treatment is not required, but footwear must be selected with care to obtain adequate width In moderate cases sufficient relief is often afforded by regular chiropody, protection of the bunion with pads of felt, and sometimes by wearing a rubber wedge between the great and second toes to reduce the deformity. In severe cases operation is advised. The

blood stream or through a penetrating wound. The joint surfaces are destroyed and the cavity is distended with pus.

Clinical features. There is acute local pain, with swelling and redness over the joint. Movements are limited and painful. There is often some degree of pyrexia.

Diagnosis. When one of the joints of the great toe is affected care must be taken to distinguish the condition from gout.

Treatment. Antibiotic drugs are given systemically. The joint is drained and rested until the infection is overcome.

OSTEOARTHRITIS OF THE TOE JOINTS

In practice, osteoarthritis in the toes is seen commonly only in the metatarso-phalangeal joint of the great toe. This has been termed *hallux rigidus*. Occasionally the metatarso-phalangeal joint of one of the smaller toes is affected, usually as a late result of osteochondritis of the metatarsal head.

FIG. 294

FIG. 295

Hallux rigidus. Figure 294 shows the marked impairment of dorsiflexion. Figure 295 shows the radiographic changes typical of osteoarthritis.

HALLUX RIGIDUS

This is osteoarthritis of the metatarso-phalangeal joint of the great toe Like osteoarthritis elsewhere, it is caused by wear-and-tear, but previous injury or disease of the joint is an important predisposing factor.

Pathology. The affected joint is sharply angled into flexion. Secondary contracture of the plantar aspect of the joint capsule fixes the deformity, and a callosity usually forms over the dorsum of the flexed joint, from pressure against the shoe.

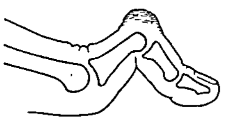

FIG. 292

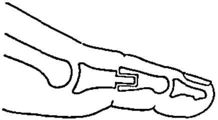

FIG 293

Figure 292—Hammer toe. The typical deformity, with callosity over the prominent proximal joint. Figure 293—Spike arthrodesis, for correction of hammer toe.

Clinical features. Typically the deformity affects only one toe—usually the second. In the characteristic deformity the proximal interphalangeal joint is in fixed flexion, and the distal interphalangeal joint, though still mobile, rests in compensatory hyperextension (Fig. 292). The symptoms, if any, are caused by the overlying callosity.

Treatment. If symptoms are slight the deformity may be accepted, or conservative treatment by protective felt pads may be sufficient In severe cases operation gives gratifying results. The joint surfaces are excised and the joint is arthrodesed in the straight position by the " spike " technique shown in Figure 293.

UNDER-RIDING TOE

Children are often brought up by their parents because one of the smaller toes, often the fourth, is curled inwards and lies beneath the adjacent toe. The condition causes no symptoms during childhood, and it should be left alone until adolescence or early adult life, when the toe can easily be straightened if it begins to cause symptoms. Similar principles apply to the management of over-riding toe.

PYOGENIC ARTHRITIS OF A TOE JOINT

Pyogenic arthritis may affect any of the toe joints, but it is uncommon The organisms may reach the joint through the

blood stream or through a penetrating wound. The joint surfaces are destroyed and the cavity is distended with pus.

Clinical features. There is acute local pain, with swelling and redness over the joint. Movements are limited and painful. There is often some degree of pyrexia.

Diagnosis. When one of the joints of the great toe is affected care must be taken to distinguish the condition from gout.

Treatment. Antibiotic drugs are given systemically. The joint is drained and rested until the infection is overcome.

OSTEOARTHRITIS OF THE TOE JOINTS

In practice, osteoarthritis in the toes is seen commonly only in the metatarso-phalangeal joint of the great toe. This has been termed *hallux rigidus*. Occasionally the metatarso-phalangeal joint of one of the smaller toes is affected, usually as a late result of osteochondritis of the metatarsal head.

FIG. 294

FIG. 295

Hallux rigidus Figure 294 shows the marked impairment of dorsiflexion. Figure 295 shows the radiographic changes typical of osteoarthritis.

HALLUX RIGIDUS

This is osteoarthritis of the metatarso-phalangeal joint of the great toe. Like osteoarthritis elsewhere, it is caused by wear-and-tear, but previous injury or disease of the joint is an important predisposing factor.

Pathology. The changes are like those of osteoarthritis in other joints. The articular cartilage is gradually worn away from both surfaces of the joint until eventually the subchondral bone is exposed. The exposed bone becomes hard and glossy (eburnation). The marginal bone hypertrophies to form osteophytes, which often form a considerable excrescence, especially at the dorsum of the toe.

Clinical features. The complaint is of pain in the base of the great toe on walking. *On examination* the metatarso-phalangeal joint is palpably thickened from osteophyte formation. If an osteophyte is especially prominent on the dorsal or medial aspect of the joint a thick-walled bursa (" bunion ") may form over it; occasionally the bursa is distended with fluid. Flexion and extension of the toe at the metatarso-phalangeal joint are restricted —usually markedly so by the time the patient seeks advice (Fig. 294). Forced dorsiflexion of the painful joint on walking is the main source of the disability.[1] *Radiographs* confirm the presence of osteoarthritis. The cartilage space is narrowed, the subchondral bone tends to be sclerotic, and there is spurring or " lipping " of the joint margins (Fig. 295).

Treatment. In mild cases treatment is not required. When treatment is called for, conservative measures are usually worth a trial first. A metatarsal bar should be fitted beneath the sole of the foot at the metatarso-phalangeal level. This acts as a " rocker " in walking, and so reduces the dorsiflexion required of the toes when weight is brought on to the forefoot from the heel. A course of short-wave diathermy to the joint may also be helpful.

When the disability is severe operative treatment should be advised. The most satisfactory method is to create a flail joint by excision of the base of the proximal phalanx (Keller's arthroplasty), as for hallux valgus. Arthrodesis of the joint is an alternative operation

OSTEOARTHRITIS OF OTHER TOE JOINTS

Osteoarthritis of the other metatarso-phalangeal joints is uncommon except as a sequel to Freiberg's osteochondritis of a metatarsal head (p. 408)

[1] It should be remembered that the normal range of dorsiflexion at the metatarso-phalangeal joint of the great toe is nearly 90 degrees

GOUTY ARTHRITIS OF THE GREAT TOE JOINTS

(General description of gouty arthritis, p. 44.)

The joints of the great toe are those most often affected in gout, especially in the first attack.

Cause. The precise cause of gout is unknown. A predisposition to the disease may be inherited. In those who are susceptible, the drinking of large quantities of beer or heavy wines or the eating of large quantities of meat is thought to be an important factor in bringing about an attack. Injury may also precipitate an attack.

Pathology. There is a disorder of purine metabolism. The primary factor is probably an impaired ability of the kidneys to excrete uric acid. In consequence, the uric acid content of the blood is increased from the normal 3.5 mg. per cent (Folin) sometimes to as much as 6 mg. per cent. An " attack " of gout occurs when the uric acid is deposited, as sodium biurate, in the cartilage of the joint.

Clinical features. The patient is usually over 40, and more often a man than a woman. There is a sudden onset (often during the night) of severe pain in the great toe. *On examination* the toe is swollen, red, and extremely tender. Joint movements are impossible because of pain There is sometimes slight pyrexia. The patient will usually recall previous similar attacks lasting a few days, with freedom from pain in the intervals. *Radiographs* are normal in the early stages. *Investigations :* There is sometimes a mild leucocytosis. The blood uric acid level is high immediately before an attack ; it lessens soon after the onset of symptoms, and gradually rises again as the attack subsides. Aspiration of the joint may yield clear fluid, but never pus.

Chronic gout In this form deposits of sodium biurate in and around the joints of the great toe lead to persistent nodular thickening *Radiographs* show rounded areas of transradiance in the bone ends, these represent deposits of sodium biurate (which is transradiant) in the subchondral bone.

Diagnosis. Acute gout is easily mistaken for acute infective arthritis Features suggestive of gout are : a raised blood uric acid level ; a history of previous attacks, with symptom-free intervals, the presence of tophi in the ears or elsewhere ; and a

good response to colchicum. Leucocytosis, if present at all, is much less marked than it is in acute infective arthritis. If doubt still exists, the joint should be aspirated : in gout aspiration may yield clear sterile fluid, but never pus.

Course. Gout usually occurs in recurrent attacks. In the early years, an acute attack subsides within a few days, leaving the joint clinically normal. In chronic gout the joint is gradually disorganised, causing permanent disability.

Treatment. Acute attacks respond to normal doses of colchicum by mouth, or to phenyl-butazone (Butazolidin). Meanwhile the foot should be rested, and the toe should be protected from pressure by a bulky wool dressing and bandage. Adjustments should be made to the diet in an effort to prevent or reduce further attacks (p. 46).

OSTEOCHONDRITIS OF A METATARSAL HEAD

(Freiberg's disease)

This is a further example of osteochondritis, an affection of developing bony nuclei. The common sites at which osteo-

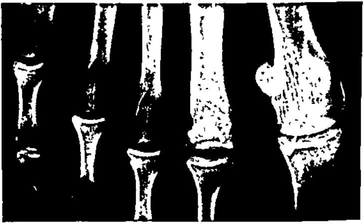

FIG 296

Freiberg's osteochondritis of the second metatarsal head
Note the fragmentation and the square-shaped deformity

chondritis occurs were listed on p. 77. The essential feature of Freiberg's disease is temporary softening of a metatarsal

head, which may become deformed under the pressure of weight-bearing.

Pathology. The epiphysis of one of the metatarsal heads—nearly always the second or third—is the part affected. The bony nucleus becomes soft and granular. While in this state it is crushed by the pressure against it of the base of the proximal phalanx of the toe. The articular surface of the metatarsal head thus loses its normal dome-shaped contour and becomes flat (Fig. 296). After about two years the texture of the bone returns to normal, but flattening of the articular surface remains. Later, the distortion of the joint surface often leads to osteoarthritis.

Clinical features. At the time of onset the patient is 14 to 18 years old. There is pain in the affected metatarso-phalangeal joint, worse on standing or walking. *On examination* there is slight thickening in the region of the head of the metatarsal, which is tender on pressure. Movements of the metatarso-phalangeal joint are slightly restricted and painful. *Radiographs* reveal the nature of the trouble. At first the epiphysis of the metatarsal appears fragmented, with patches of increased density. Later, the articular surface is flattened, so that the end of the bone appears square-cut instead of round (Fig. 296).

Treatment. Since the symptoms are relatively slight treatment is not essential. Sometimes it is justifiable to rest the joint by immobilising the foot in a walking plaster for two months. This is usually sufficient to relieve most of the pain. If troublesome osteoarthritis develops later, the head of the metatarsal should be excised.

INGROWING TOE NAIL

Ingrowing toe nail is common only in the big toe.

Cause. Some persons have toes that are particularly prone to ingrowing nail. Incorrect cutting of the nail is a contributory factor (Fig 298).

Pathology. A sharp anterior corner of the nail impinges against the skin fold. The mechanical irritation is followed by bacterial infection of the skin fold. The lesion may be at the medial or lateral border of the nail, or both.

Clinical features. There is pain at the affected corner of the

nail. *On examination* the skin fold is inflamed, and there may be local suppuration where the corner of the nail digs into the skin.

Treatment. In mild and early cases conservative treatment may suffice. Pledgets of gauze soaked in surgical spirit are tucked beneath the corner of the nail twice a day, and the nail is allowed to grow until its edges project beyond the skin folds (Fig. 297).

FIG. 297 FIG. 298 FIG 299

Figure 297—Nail cut correctly. Figure 298—Nail cut too short: corners dig into pulp. Figure 299—Wedge operation for ingrowing toe nail

In long-standing cases operation is advisable. The simplest method is to excise a wedge of the nail bed together with the margin of the skin fold (Fig. 299).

In the worst cases permanent ablation of the nail by excising the nail bed is more satisfactory. It is unnecessary to shorten the toe to obtain adequate skin cover; the raw surface can be covered by advancing a dorsal skin flap, or, if necessary, by a free skin graft.

SUBUNGUAL EXOSTOSIS

A subungual exostosis is a bony outgrowth from the dorsal surface of the distal phalanx of a toe—usually the great toe It projects upwards and forwards between the tip of the nail and the terminal pulp. The nail is raised and deformed, and the skin of the pulp overlying the outgrowth is thickened and hard There is severe pain when pressure is applied over the nail or terminal pulp. *Radiographs* show the exostosis, which is seen best in the lateral projection

Treatment. The exostosis should be excised through a terminal incision just beyond the tip of the nail.

ONYCHOGRYPOSIS

Translated from the Greek, this means "hooked nail." The term is descriptive. The nail—usually of the big toe—is enormously thickened and curved, eventually resembling a miniature ox-horn.

Treatment. Simple removal of the nail is an adequate temporary measure; but the new nail *will* become similarly deformed. *For* permanent cure the nail bed must be excised.

nail. *On examination* the skin fold is inflamed, and there may be local suppuration where the corner of the nail digs into the skin.

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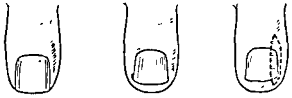

Fig. 297 Fig. 298 Fig. 299

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Index

D

E

F

I

J

K

L

P

Q

R